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TRAUMATIC THORACIC ANEURYSMS: TREATMENT BY RESECTION AND GRAFTING WITH THE USE OF AN EXTRACORPOREAL BYPASS

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IN THE past 2 centuries a large number of examples of traumatic aneurysms of the major vessels have been recorded. They result from direct and indirect force, the latter being identified as soon as necropsies were conducted frequently. Among the earliest instances were those associated with falls,[19, 22, 25] direct trauma to the chest (frequently from a horse kick),[2, 5, 10] and a variety of transportation injuries.[12, 16, 20, 24, 29] With the advent of rapid transportation the frequency has steadily increased, so that at present we are faced with many decelerative injuries, among which are those affecting the thoracic aorta. It is the purpose of this paper to report 4 cases of traumatic thoracic aneurysm which were resected with the aid of an extracorporeal bypass.

ACCELERATION AND DECELERATION AORTIC INJURIES

Deceleration injuries occur most commonly to passengers in automobile or airplane accidents, whereas acceleration injuries usually involve pedestrians who are struck by fast-moving vehicles. Insofar as the thoracic aorta is concerned the resulting lesions are the same.

The most frequent site of rupture of the thoracic aorta is that part immediately distal to the ligamentum arteriosum. Of 72 cases collected by Strassman,[28] 40 were at that level, 14 were in the ascending aorta just beyond the aortic valve, and the remainder were in the descending thoracic and abdominal aorta. Traumatic rupture of the abdominal aorta occurred in only 3 cases.

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TABLE I. CASES OF TRAUMATIC ANEURYSM TREATED BY EXCISION

AUTHOR	OPERATION DATE	AGE	DURATION AND CAUSE	SITE	OPERATION	ANCILLARY TECHNIQUE	OCCLUSION (MINUTES)	RESULTS
Bahnson[3]	1952	49	2 yr. Auto	Ligamentum arteriosum	Endoaneurysmorrhaphy	Nil	7	Successful
Johnston[17]	1953	66	3 yr. Auto	Ligamentum arteriosum	Lateral excision	Nil	0	Successful
Stranahan[27]	1953	25	5 yr. Auto	Ligamentum arteriosum	Excision and homograft	Left subclavian artery and descending aorta, Tygon shunt	200	Successful
Hardin[14]	1953	32	10 yr. Struck by coal conveyor	Ligamentum arteriosum	Excision and homograft	Hypothermia	41	Ventricular fibrillation
Goyette[11]	1953	23	4½ yr. Auto	Ligamentum arteriosum	Lateral excision and polythene wrap	Nil	0	Died 3 mo. from rupture of sac
Cooley[7]	1954	31	9 yr. Crush	Arch and upper thoracic aorta	Excision and homograft	Hypothermia	62	Successful
Julian[18]	1954	22	5 yr. Auto	Ligamentum arteriosum	Excision and homograft	Hypothermia and hypotension	30	Successful
Eiseman[9]	1954	32	10 yr. Gunshot wound	T. 10 and 11	Excision and homograft	Hypothermia	63	Paraplegia
Ware[30]	1954	19	9 wk. Auto	Ligamentum arteriosum	Excision and homograft	Shunt and hypotension	210	Died; graft leaked as result of esophagopleural fistula at 7 days
Adams[1]	1955	30	1 yr. Crush	Ligamentum arteriosum	Excision and homograft from left subclavian artery to descending aorta	Definitive graft used as shunt	0	Successful
Storey[26]	1955	35	4 yr. 20 ft. fall	Ligamentum arteriosum	Excision and homograft	Left subclavian artery to descending aorta, pig aorta shunt, and hypotension	?	Successful
Hardin[13]	1955	31	12 yr. Landmine	Descending aorta, multiple	Excision and orlon graft	Hypothermia	50	Successful; proximal B.P. rose from 60 to 120 systolic
Cooley[6]	1956	50	25 yr. Horse kick	Ascending aorta	Excision and homograft	Pump oxygenator	31	Successful; right carotid perfused 200 c.c./min.
Gerbode	1956	18	2 yr. Auto	Ligamentum arteriosum	Excision and homograft	Pump; shunt left atrium to distal aorta	50	Died from postoperative hemorrhage
Gerbode	1956	25	13 yr. Auto	Ligamentum arteriosum	Excision and homograft	Pump; shunt left atrium to femoral artery	33	Successful
Gerbode	1957	19	3 yr. Auto	Ligamentum arteriosum	Excision and homograft	Pump; shunt left atrium to femoral artery	45	Successful
Gerbode	1957	23	2 mo. Auto	Ligamentum arteriosum (ruptured)	Excision and homograft	Pump; shunt left atrium to femoral artery	57	Successful

The fixation of the aorta accounts for the sites of most frequent tear.[15] The transverse arch is held by the innominate, carotid, and subclavian vessels. The support is further increased by the ligamentum arteriosum, which is attached to the pulmonary artery. It is also fixed at the diaphragm. Between the left subclavian artery and the diaphragm only pleura and intercostal arteries retard the forward snap of the thoracic aorta in rapid deceleration.[23] The blood-filled thoracic aorta acts like a solid viscus, adding weight, and therefore force, to the forward motion. Rupture above the aortic cusps may occur in deceleration injuries because the heart continues to move forward while the arch remains fixed.

The injuries may vary from intimal tears, which are usually harmless, to complete severance, which results in death. Tears in the intima and media, with retention of the adventitia and pleura, cause the saccular aneurysms which are the common finding in those who survive the immediate injury.

Diagnosis.—Symptoms may be entirely absent, as indeed may be the external evidence of thoracic injury. Pain in the chest is common and it may be associated with symptoms of compression of the bronchus or recurrent laryngeal nerve. Roentgen examination is diagnostic and frequently will present conclusive evidence of the aneurysm without signs or symptoms; widening of the mediastinum immediately after the injury is a characteristic early sign. Angiography may be necessary if there is doubt.

Treatment.—At present all the former methods of treating thoracic aortic aneurysms have given way to resection and grafting whenever possible. This has been shown to be feasible even for those affecting the ascending aorta, although some form of extracorporeal circulation is necessary to accomplish this.[6] For those traumatic aneurysms involving the descending aorta, a variety of methods have been used to preserve the viability of the spinal cord and the viscera during aortic cross-clamping. Intraluminal shunts make suturing difficult and necessitate at least some period of total occlusion. Bypass grafts in which the proximal and distal aorta are temporarily connected with a shunt have been used with success, but they are tedious to construct and at times cannot be used at all because of the anatomic limitations of the aneurysm itself. Hypothermia permits one to cross-clamp the aorta for a longer period of time, but left ventricular strain and proximal hypertension are sometimes difficult to control and may lead to ventricular fibrillation.

Traumatic aneurysms that have been treated by excision with the aid of various ancillary techniques are summarized in Table I.

As a consequence of experiments in aortic cross-clamping conducted in our laboratory during the past year it was concluded that one could safely cross-clamp the canine thoracic aorta for 90 minutes without spinal cord damage, with a flow of only 15 c.c. per kilogram of blood passing directly from the brachial artery to the femoral or from the left atrium to the femoral artery by means of a rotary pump.[4] The latter technique proved to have the advantage of making it possible to control proximal hypertension and left ventricular strain because

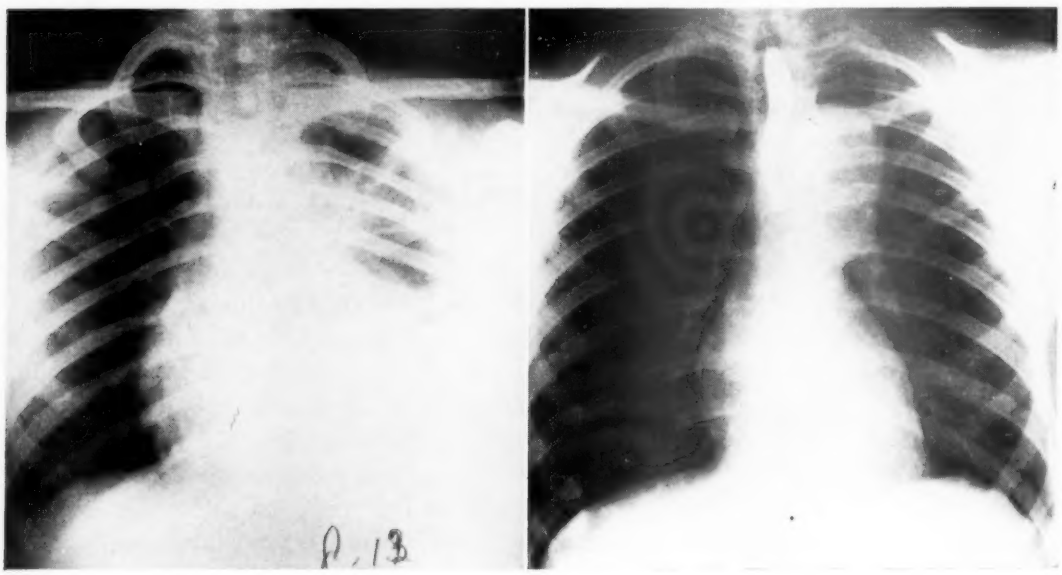
of the greater flow. Variations in proximal blood pressure are common, making it necessary to monitor this continuously. The technique has also been used by DeBakey and Cooley.[8, 21]

The 4 instances of thoracic aortic aneurysms reported in this paper resulted from automobile accidents: one from rapid acceleration, and 3 from rapid deceleration. All were resected with the aid of an extracorporeal pumping system.

CASE REPORTS

CASE 1.—T. S., an 18-year-old white man, had been hit by an automobile 2 years before admission, resulting in bilateral fractures of the lower legs, a right pneumothorax, and left hemothorax (Fig. 1, *A*).

On entry to the hospital he was asymptomatic and leading a normal life. There was no shortness of breath, difficulty with eating nor thoracic pain. There were prominent pulsations in the neck above the anterior third of the left clavicle, and a Grade II systolic murmur could be heard over the upper left thorax. No other abnormality was detectable. Fluoroscopy revealed what appeared to be a saccular aneurysm lying in the left upper thorax in close association with the left half of the arch and first portion of the descending aorta; its exact origin was undetermined (Fig. 1, *B*). Laboratory studies were normal; a previous Wassermann was negative.

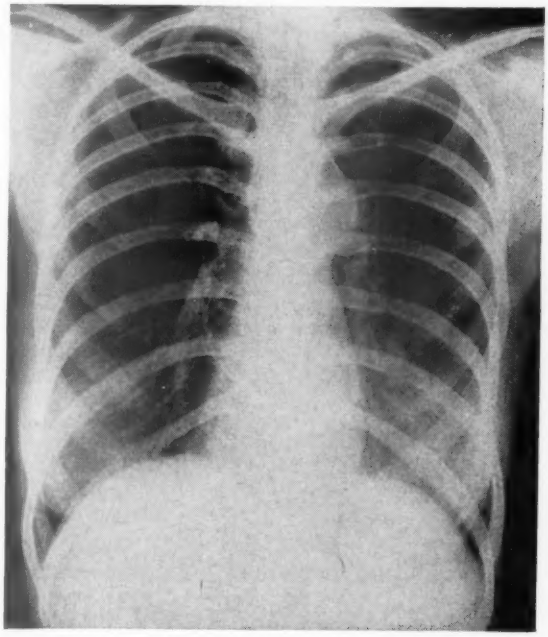


A. *B.*

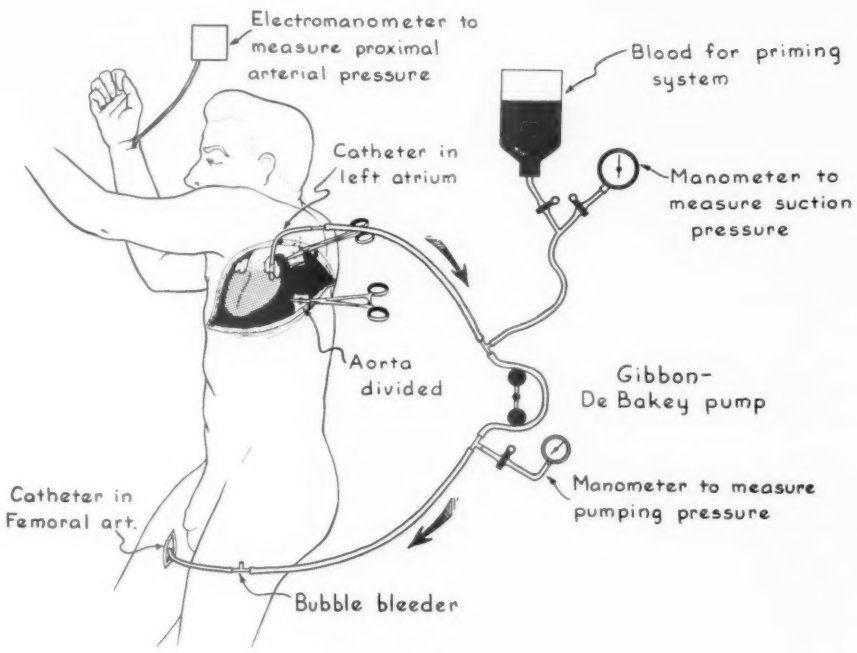
Fig. 1.—Case 1. *A*, Roentgenogram taken shortly after injury. Note the mediastinal widening. *B*, Roentgenogram taken 18 months after injury.

On Oct. 18, 1956, operation was performed under general anesthesia through an incision in the bed of the fifth left rib. The pleural space was completely obliterated by dense scar tissue. Considerable time and care were required for mobilization of the lung in order to gain access to the aorta. A pulsating aneurysm approximately the size of an orange was exposed just distal to the left subclavian artery and involving its origin to a certain extent. Distally the aorta was enveloped in somewhat scarred pleura but was pulsating normally. It was friable, as though it had lost some of its elastic properties, or perhaps had been dissected. The aorta and aneurysm were mobilized with some difficulty because of perivascular reaction; there were dense adhesions medial to the pulmonary artery, posterior to the bronchus, and superior to the mediastinal tissues. The distal aorta was partially occluded while an Ivalon shunt approximately 1 cm. in diameter was inserted end-to-side.

After the patient had been given 1 mg. of heparin per kilogram of body weight, a catheter was placed in the left auricular appendage through a purse-string suture and connected by polyvinyl tubing through a Gibbon-DeBakey roller pump to the distal aortic Ivalon shunt. The aneurysm was excised and replaced by a homograft 4 inches in length. During the excision the pump was run at a rate of 700 to 1,000 c.c. per minute, which maintained a



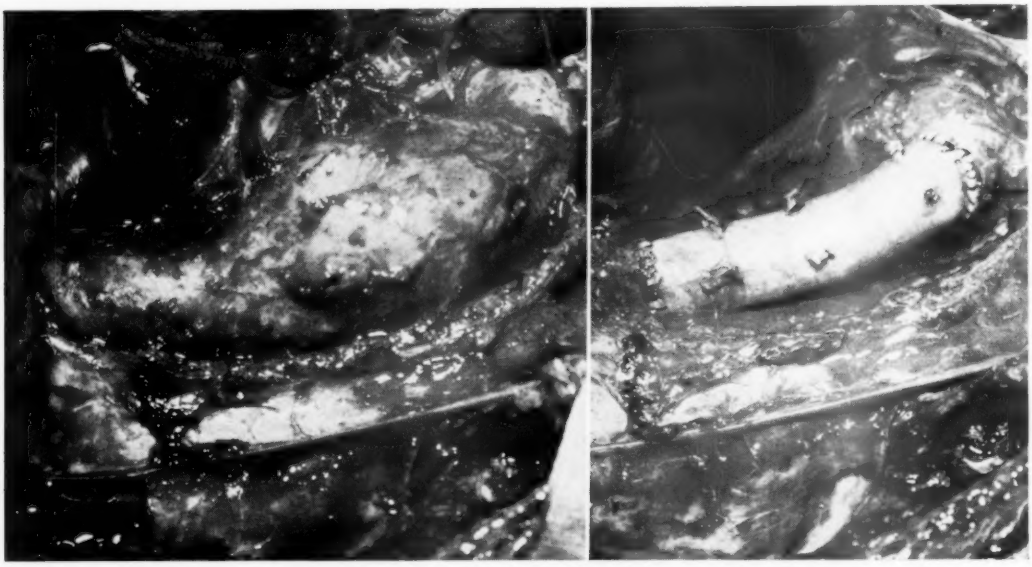
A.



B.

Fig. 2.—Case 2. *A*, Roentgenogram taken 13 years after injury. *B*, Diagram of extracorporeal bypass. A Bowman pump is used to keep the proximal arterial pressure catheter patent. (*C*, *D*, and *E* on following page.)

pressure of 40 to 90 mm. Hg in the distal aorta for the period of bypass, which lasted 50 minutes. The wound oozed rather freely during this time, probably due to freeing of the extensive adhesions from the lung, but conditions were felt to be satisfactory and the wound was closed. Protamine was given to reduce the clotting time to normal. The patient was returned to his room in fair condition with good pedal pulsations, and upon awakening he apparently had no neurological deficit.



C. *D.*

E.

Fig. 2.—contd.—*C*, The aneurysm at operation. *D*, The graft in position. *E*, Postoperative roentgenogram.

The patient went into shock 7 hours after the completion of the operation and was given further transfusions. Despite resuscitative measures he expired.

At necropsy there was a considerable amount of blood in the pleural cavities and atelectasis of the left lung. The inserted graft in the descending aorta was intact without extensive surrounding hemorrhage. No gross leakage had occurred. The hemorrhage apparently had come from the numerous divided adhesions.

Grossly the removed segment of thoracic aorta showed an aneurysmal dilatation which measured up to 11 cm. in maximal diameter. The aortic wall was of varying thickness with irregularly roughened, thickened intima. Microscopically the intima appeared irregularly thick and fibrous, with a few fragments of thrombus upon its surface. Small remnants of elastic tissue were visible in the aneurysmal wall.

Comment.—The main complicating factor in this case was the presence of the large numbers of pleural adhesions, which made the dissection tedious and time-consuming and caused the loss of a considerable amount of blood, particularly when heparin was used. The exact site of the bleeding was impossible to define at necropsy. In retrospect it might have been wise, in spite of the poor condition of the patient, to return him to the operating room and attempt to stop some of the extensive oozing, but at the time it was felt that his general condition precluded this.

CASE 2.—D. G., a 25-year-old white woman, had been in an automobile accident at the age of 12 at which time she sustained a massive total body injury, was unconscious for one day, and had fractures of the left femur and several ribs.

She complained of fever, productive cough, shortness of breath, pain in the left chest, and a 20-pound weight loss. Physical examination revealed scattered râles throughout the left lung with inconstant wheezing. The remainder of the examination was negative. Chest roentgenograms revealed a calcified enlargement of the mediastinum protruding to the left, which was interpreted as being an aortic aneurysm. Bronchoscopy revealed 50 per cent of the left main bronchus in its mid-point. Exploratory thoracotomy was performed at the U. S. Naval Hospital, Oakland, Calif., on March 24, 1954. An aneurysm of the descending aorta was found and the thorax was closed without definitive surgery.

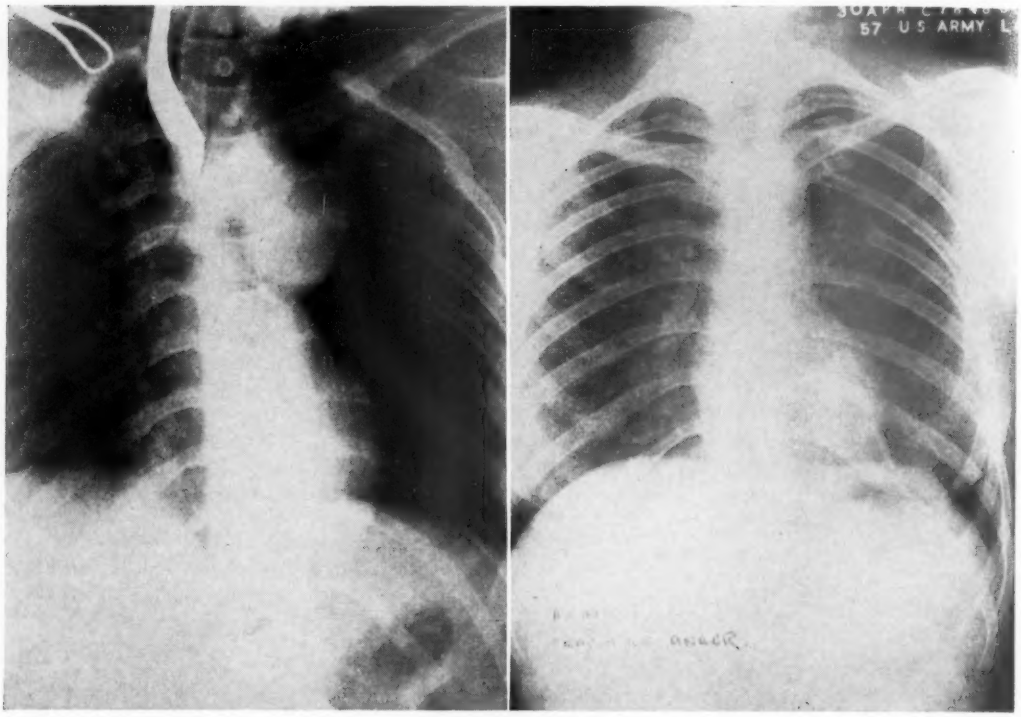
Postoperatively she developed increasing symptoms of shortness of breath and of increasing difficulty with sputum and chronic infection in the left lung (Fig. 2, *A*).

Because of the known prognosis of aortic aneurysms, the patient was again operated upon on Dec. 13, 1956. The thorax was opened widely through the fifth intercostal space and the aorta was mobilized. A plastic catheter was inserted into the left auricular appendage and connected to a plastic tube leading through a Gibbon-DeBakey pump to a metal cannula in the right femoral artery (Fig. 2, *B*). The bypass was begun at the rate of approximately 500 c.c. per minute. The aneurysm (Fig. 2, *C*) was excised and a freeze-dried aortic homograft was sutured into place (Fig. 2, *D*). The occlusion time was 33 minutes. There was no evidence of renal damage from the aortic clamping and bypass; urinary output stopped during the procedure, but was resumed immediately upon release of the aortic clamps.

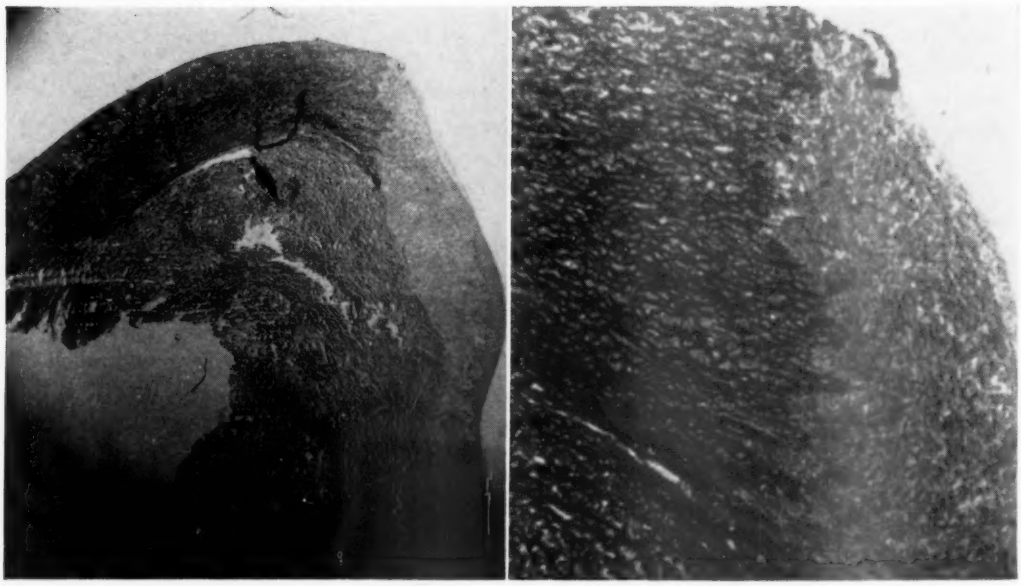
The postoperative course was uneventful, with no depression of urinary output and no neurological deficit. She was discharged on the twelfth postoperative day. She is doing well now and has no pain. Her cough has disappeared (Fig. 2, *E*).

CASE 3.—M. M., a 19-year-old white woman was admitted to Stanford University Hospital on Jan. 6, 1957. A mediastinal mass had been found on a routine roentgenogram in September, 1956, and diagnostic studies, including arterial visualization (Fig. 3, *A*), had confirmed the presence of an aortic aneurysm. Laboratory studies, including CBC, urinalysis, and serology, were normal. Her past history revealed that she had been involved in an automobile accident in 1954, when her left wrist was fractured, considerable pressure was exerted upon her chest, and she was unconscious for 2 days.

On examination the heart was not enlarged. There was a fairly loud systolic murmur heard best along the left sternal border when the patient lay flat. No other abnormality was



A. *B.*



C. *D.*

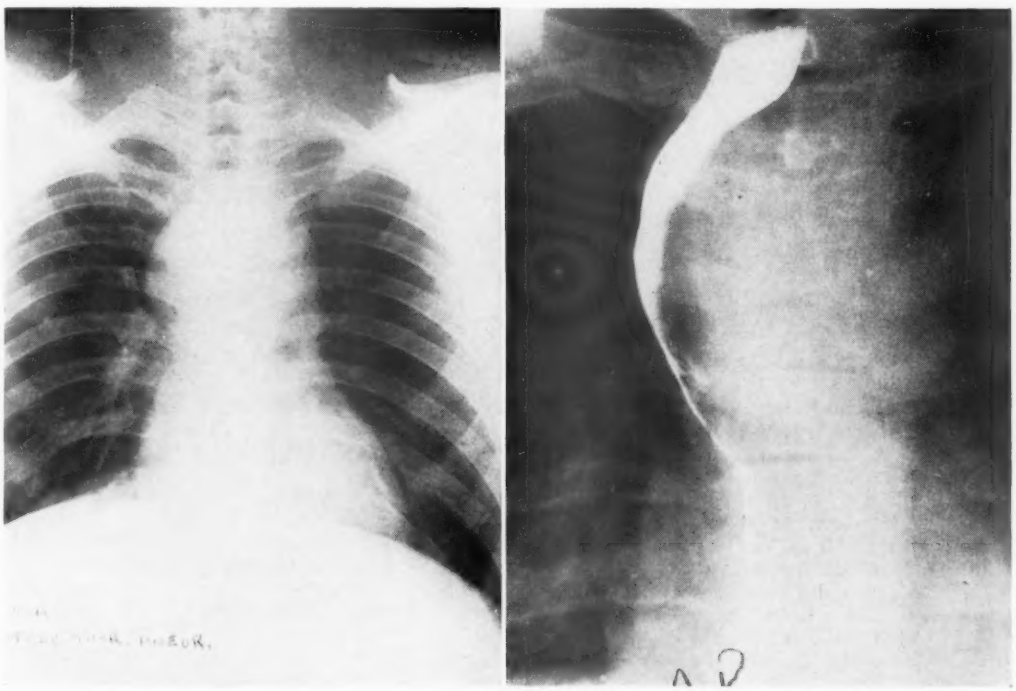
Fig. 3.—Case 3. *A*, Preoperative angiogram. *B*, Postoperative roentgenogram. *C*, Wall of traumatic aneurysm showing abrupt transition from normal media to wall composed only of fibrous tissue and thickened periadventitial connective tissue. *D*, High-powered view of *C* depicting zone of abrupt transition from media, showing irregularly fragmented margin of fibrous lining of traumatic aneurysm.

found. Fluoroscopy of the chest showed a well-localized bulging in the distal portion of the aortic arch, with some linear calcification. The heart was not enlarged and the lungs appeared clear.

On Jan. 8, 1957, operation was performed. The thorax was opened through the bed of the fifth left rib. There was a free pleural space except for some adhesions near the upper lobe, which were cut. The aortic aneurysm was then exposed, lying just beyond the left subclavian artery, with about 1.5 cm. of normal tissue between the lower border of the left subclavian artery and the margin of the aneurysm, which was about 6 cm. in diameter and 7 cm. in length. The aneurysm was freed from the surrounding tissues. It was necessary to ligate 2 pairs of intercostal arteries to obtain adequate exposure of the distal end. Extracorporeal circulation was set up as before after the patient had been heparinized with 1 mg. of heparin per kilogram of body weight.

A proximal pressure of approximately 130 mm. Hg systolic was maintained after the clamps were applied, with a flow of about 1 liter per minute for the 45 minutes of the bypass, maintaining a pressure of about 50 to 70 mm. Hg in the femoral artery. The aneurysm was excised and a thoracic homograft was interposed. The patient withstood the operation extremely well and had hardly any change in pulse rate throughout the procedure. The platelet count fell from 340,000 to 265,000 during the bypass.

Postoperatively the patient had a temporary right upper lobe collapse, but recovery was otherwise uninterrupted and there was no neurological or renal deficit (Fig. 3, *B*).



A. *B.*

Fig. 4.—Case 4. *A*, Preoperative roentgenogram. *B*, Preoperative barium swallow roentgenogram, anteroposterior view.

On gross pathologic examination the specimen showed an aneurysmal sac, 4 cm. in diameter, connecting the separated ends of the aorta. The wall of the sac was thin, greyish white, and contained yellowish plaques and zones of calcification. The aortic walls were thickened and yellow. Microscopically the wall of the aneurysm was composed of dense fibrous tissue devoid of elastic fibers, with plaques of eosinophilic amorphous tissue and flecks of calcification (Fig. 3, *C* and *D*).

CASE 4.—M. A., a 23-year-old white man, had been involved in an automobile accident on March 14, 1957, when he suffered a fractured skull, facial lacerations, and chest injuries, and was unconscious for 5 days. Subsequently he had frequent episodes of chest pain and blood was aspirated from the pleural space on several occasions. He had difficulty in breathing and swallowing, and latterly episodes of nausea and vomiting. He had been completely asymptomatic before the accident.

On examination one month later there was a blowing systolic murmur at the left sternal border transmitted to the left shoulder but not to the back. Chest films showed that the esophagus was deviated to the right by a mass in the superior mediastinum (Fig. 4, *A* and *B*).

Laboratory studies were essentially normal.

Operation was performed on April 29, 1957. The thorax was opened through the bed of the fifth rib, which was excised. An aneurysm of the thoracic aorta the size of a grapefruit just distal to the subclavian artery was seen. The major vessels were mobilized and extracorporeal circulation was set up as in the previous 2 cases. After the proximal and distal vessels were clamped, the aneurysm was opened and it was seen then that its medial part had ruptured and that there was a large clot partially encircling the trachea and esophagus. The aneurysm was excised and replaced with a homograft 4 inches in length. The bypass lasted 57 minutes. The flow rate was 800 c.c. per minute which maintained an average pressure of about 70 mm. Hg in the distal aorta.

Six hours postoperatively he developed leakage from one of the suture lines in the area which we had been unable to cover, necessitating his return to the operating room where the bleeding was successfully controlled. He subsequently made an uninterrupted recovery.

Pathologic studies of the aneurysmal wall showed a thick zone of organized cellular fibrous tissue. An abrupt junction was seen where it joined the normal aorta.

SUMMARY

Four cases of traumatic thoracic aneurysm have been treated by excision and homograft replacement, with the aid of extracorporeal bypass circulation. One patient died postoperatively from hemorrhage; the other 3 had satisfactory recoveries. No renal or neurological deficits were noted postoperatively in the 4 cases.

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PROBLEMS RELATED TO A BUBBLE OXYGENATOR SYSTEM

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THE bubble oxygenator is a simple extracorporeal device which performs well only when certain features peculiar to its operation are understood. Our experience in overcoming the problems related to the proper functioning of the bubble oxygenator may be useful to those interested in the clinical application of this type of perfusion apparatus.

Experimental and clinical data suggest that high blood flow rates are desirable during total body perfusion. When bubble oxygenators are employed at high blood flow rates, the hazards of air emboli become correspondingly greater. A common problem in the successful operation of any bubble oxygenator is the prevention of air emboli in the arterial system. The solution to this problem in our hands depended upon the recognition of the importance of blood reservoir levels in the oxygenator. Another major defect of the bubble oxygenator is its tendency to produce foam which can result whenever oxygen is bubbled through blood. If foam is allowed to become static, fibrin is formed and the resulting fibrin strands may also serve as a matrix for deposition of platelets from the blood. An additional damaging effect of foam is the denaturation of blood proteins. The use of antifoaming agents has been of great benefit in decreasing the production of foam in bubble oxygenators, but these agents have not completely eliminated the hazard of static foam. The adoption of plastic mesh to disperse foam and larger air bubbles has eliminated the formation of fibrin in our oxygenator.

Increased safety in operation of the extracorporeal system has been gained by the addition of several mechanical refinements. These refinements included a plastic bubble trap, a blood filter, and connectors for tubing which prevent separation of the tubing during a perfusion.

The direct relation between the use of barbiturates and survival rates of animals in the laboratory has become apparent. Recognition of this problem and the substitution of different anesthesia agents has consistently improved the results of experimental cardiac by-pass.

DESCRIPTION OF THE BUBBLE OXYGENATOR

The oxygenating unit is constructed of Lucite and has been adapted from a number of oxygenators evolved by others. The oxygenator (Fig. 1) most

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*Fellow, Michigan Heart Association.

nearly resembles that developed by Nelson.[8] It is a cylinder within a cylinder. The inner cylinder is the oxygenating column; it measures 5 cm. in diameter and 41 cm. in length. The outer cylinder, which acts as the blood reservoir, is 13 cm. in diameter and 50 cm. in length. The outer cylinder has a plastic perforated screw cover for the top and is supported by a metal stand.

Oxygen is delivered to the inner column at its base through a Lucite tube which has 20 holes bored in its distal 3 cm. The holes are 0.8 mm. in diameter which is equivalent to the inside diameter of a No. 18 needle. The flow rate of oxygen is determined by the rate of blood flow required and is approximately one liter per 150 ml. blood flow per minute.

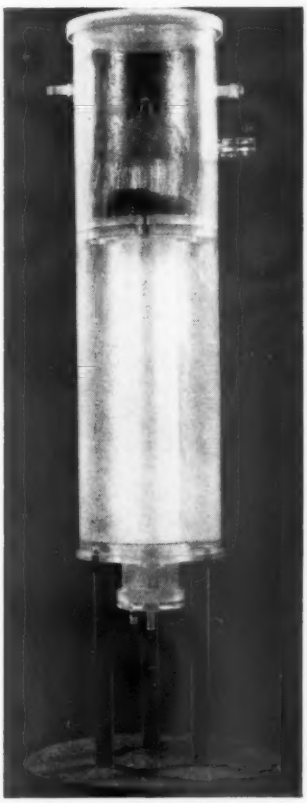

Fig. 1.—Lucite oxygenator with central oxygenating column and deep outer blood reservoir.

Venous blood enters the bottom of the inner column through a connector with an inside diameter of 7 mm. As the blood is pumped into the inner cylinder, it encounters and is mixed with rising bubbles of oxygen. When the blood reaches the top of the inner column it passes through a plastic mesh bubble dispenser coated with Antifoam. After passing through this plastic mesh cover, the blood spills onto a fine mesh filter placed in the outer column and collects beneath it in the large outer reservoir. Circulation is maintained by the Sigmamotor* pump model T 6S and the artificial ventricles which have

*Sigmamotor, Inc., Middleport, N. Y.

an inside diameter of 1.8 cm. are constructed of siliconized rubber. Tygon plastic tubing with an inside diameter of 3/8 inch is used throughout the system.

ELIMINATION OF AIR AND FIBRIN EMBOLI

When oxygen is bubbled through blood, potential air emboli result. If these emboli enter the arterial circulation, varying degrees of brain or spinal cord damage can occur. When a dog sustains a severe injury of this type, characteristic mid-brain changes are present following the perfusion. The animal exhibits spastic paralysis of the forelegs anl flacid paralysis of the hind legs and death occurs usually within 48 hours. Less severe central nervous system damage may result in a prolonged recovery period.

Fig. 2.—The plastic mesh bubble disperser can be noted covering the inner oxygenating column.

Air emboli in the cerebral vessels have been demonstrated by several techniques. At times bubbles have been seen in the vessels grossly or have been suggested in microscopic sections of the brain by empty, dilated blood vessels. Microscopic examination of arterial blood before it enters the arterial circulation has revealed bubbles of air. Other investigators have used the slit lamp or injected fluorescein[5] to outline areas of brain in which circulation is absent as a result of air emboli. At times, however, the demonstration of air in the brain is impossible and can only be inferred as a cause of death. It is in these cases that other factors such as anesthesia may be responsible for the animals' death. The problem of air emboli arising from a bubble-type oxygenator has been discussed by Gollan,[3] Clowes,[1] and Helmsworth.[4] A number of investigators have abandoned the use of bubble oxygenators because the hazard of air emboli could not be eliminated.

The elimination of air emboli from the system we have used has been accomplished simply. It was noted that a vertical column of blood in the arterial reservoir maintained at more than 8 cm. from the arterial outlet assured a satisfactory animal perfusion. This represented a volume of 700 ml. of blood in the oxygenator. At the present time, it is the practice to maintain a reservoir of at least 1,000 ml. of blood in the oxygenator. The deep reservoir provides an important safety barrier which depends on the distance that the blood air interface lies above the arterial outlet. Air bubbles which might pass into the reservoir from the central oxygenating column remain near the surface and are not drawn into the arterial circuit.

A plastic mesh sponge with interstices measuring 4 mm. by 4 mm. impregnated with Antifoam* has been added as a cap on the inner oxygenating column (Fig. 2). This simple but effective bubble disperser has eliminated bubbles from the outer reservoir. The plastic mesh has not increased hemolysis after perfusion in the laboratory.

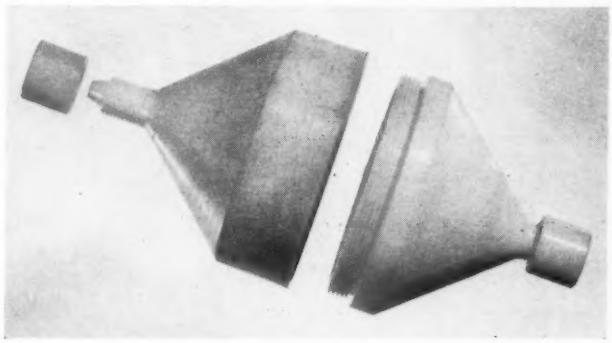

Fig. 3.—Nylon bubble trap with end nuts that lock and prevent separation of tube connections.

A varying quantity of foam results when oxygen is bubbled through blood. This increases after 20 to 30 minutes of perfusion even when the oxygenator is coated with Antifoam. Foam which becomes static deposits fibrin strands on the walls of the oxygenator. The strands of fibrin may break up and become potential emboli. These fibrin emboli are prevented from entering the arterial circuit when an adequate blood reservoir is maintained because the emboli are lighter than blood and will remain on the surface of the blood in the reservoir.

Since the plastic mesh cover has been employed to cover the inner oxygen chamber, there has been no foam in the arterial reservoir after one hour of perfusion. The prevention of foam has aided in the maintenance of clotting mechanism because fewer platelets are removed from the blood by fibrin strands. Bleeding after one hour of perfusion has not been a problem with the plastic bubble oxygenator when protamine sulfate in a dosage twice that of heparin has been administered to neutralize the heparin following perfusion.

*Dow-Corning, Midland, Mich.

BUBBLE TRAP FILTER AND SAFETY CONNECTORS

A bubble trap has not been used in animal perfusions and results have been satisfactory without it. When the oxygenator is applied clinically, however, a plastic bubble trap (Fig. 3) is placed in the arterial circuit close to the cannulated artery. The trap also functions as a receptacle for any bubbles which remain in the arterial system after all connections are completed. It is easy to milk any visible bubbles into the trap where they remain during cardiac by-pass.

A stainless steel filter (Fig. 4) with 70 × 70 mesh is placed in the outer arterial reservoir 10 cm. below the top of the oxygenating column. In this area, where no pressure is present in the system, any clot which may enter the oxygenator is prevented from passing into the arterial tubing to act as an embolus or obstruction to blood flow. When the donor blood has been prepared with 18 mg. of heparin for each 500 ml. of blood and the subject is treated with heparin 1.5 mg. per kilogram of body weight, blood clots have not been observed.

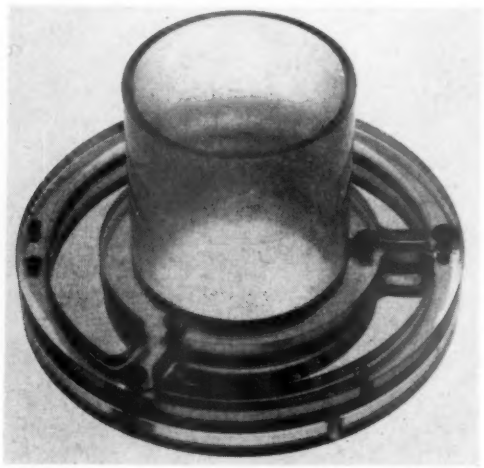

Fig. 4.—This removable blood filter slides over the inner oxygenating column and is positioned in the outer reservoir.

ANESTHESIA DEATHS IN THE LABORATORY

Death in laboratory animals is related to the method by which donor blood is prepared. The earliest donor animals were given pentobarbital. Many of the animals perfused with blood obtained in this way died or recovered only after many hours of deep anesthesia. A change to a rapidly acting barbiturate such as Surital improved the survival rate and markedly shortened the time perfused dogs remained anesthetized. At the present time the donor dogs receive succinylcholine alone and are ventilated with an endotracheal tube and mechanical respirator while blood is drawn under local anesthesia. This method of obtaining donor blood has been highly satisfactory and animals perfused with this blood recover as soon as the perfusion is completed or soon thereafter. It appears that animals believed to die as a result of

air emboli, when none can be found at autopsy, may be victims of overdosage of barbiturates. It is simple to reanesthetize a dog which has been perfused with a blood transfusion collected under barbiturate anesthesia. In these perfused dogs, the liver appears to be unable to metabolize barbiturates in a normal manner as a result of the perfusion.

FLOW RATES WITH A BUBBLE OXYGENATOR

The best results with the pump oxygenator described have been obtained with blood flow rates of 65 to 75 ml. per kilogram of body weight per minute. Kaplan[6] has indicated the narrow margin of safety present with low flow rates and his studies have influenced our application of the bubble oxygenator. Arterial perfusion rates of 65 to 75 ml. per kilogram. of body weight per minute have been satisfactory in laboratory animals with no evidence of air emboli. As experience with extracorporeal circulation has increased, blood flow rates have been increased. This trend appears to be physiologically sound. High blood flow rates are not a hazard when a blood reservoir of 1,000 ml. is present in the bubble oxygenator.

ANIMAL RESULTS

A consecutive series of 20 dogs were perfused without a mortality.[7] Perfusions ranged from 20 to 60 minutes and the plastic bubble oxygenator was used in each experiment. The oxygenator has been safe for perfusions lasting an hour and hemolysis values have not exceeded 100 mg. per cent. Platelet counts have decreased one third of their normal value after one hour of cardiac by-pass. Blood gas studies and alterations in pH have been similar to others using the bubble oxygenator.[2, 8] No attempt has been made to alter the respiratory acidosis which is seen in the postperfusion period.

SUMMARY AND CONCLUSIONS

Successful operation of a bubble oxygenator depends upon the recognition and solution of the problems inherent in this type of apparatus. A plastic bubble oxygenator employed successfully was described and the methods used to produce consistent survivals in the laboratory were discussed. The hazard of air emboli entering the arterial system was overcome by the use of a deep blood reservoir. Static foam and fibrin emboli were eliminated by placing a plastic mesh cover on the inner oxygenating column.

It was observed that dog donor blood containing barbiturates was directly related to decreased animal survival rates. The institution of a rapid muscle relaxant for blood collection improved experimental results following cardiac by-pass.

High arterial perfusion rates were employed in 20 consecutive successful dog perfusions lasting up to one hour in duration. A bubble oxygenator was utilized in all of these experimental procedures. It is concluded that blood flow rates of at least 75 ml. per kilogram of body weight per minute may be employed without danger of air emboli entering the arterial circuit.

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AN AIR TRAP FILTER FOR EXTRACORPOREAL OXYGENATOR SYSTEMS

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THE successful use of an extracorporeal oxygenator system demands that a suitable filter be installed to clear the circuit. Various authors[1-6] engaged in the problem of constructing oxygenators fail to describe the filtering mechanism. Miller and Gibbon[5] mention a filter which will evacuate air as well as remove clots but do not describe it in detail.

A filter should not only be capable of removing particulate matter, but also any bubbles which may penetrate from the blood reservoir to the arterial side of the system. This is particularly true in the use of the bubble-type oxygenator.

There are 4 necessary prerequisites before a filter may be considered adequate to insure the safety of an oxygenator. (1) It must have the ability to remove any blood clots or particulate matter. (2) It must effectively remove any bubbles of oxygen which may inadvertently enter the arterial side from the blood reservoir. (3) It must be easily inserted into the system and be simply handled by the operator. (4) It must be easily cleaned and sterilized.

These 4 rules have been incorporated into the design of a filter for use primarily with bubble-type oxygenators. However, it may be used as readily with filming types.†

DESCRIPTION OF APPARATUS

The filter consists of a screen of stainless steel mesh 60 strands per linear inch. The wire diameter is 0.007 inch and the width of the mesh opening is 0.0097 inch. The open area of the mesh is 33.9 per cent. The mesh is sealed by a rubber gasket within a body, composed of two halves, machined from acrylic plastic for cold sterilization or thermal setting plastic for autoclaving.‡ An inlet pierces one of the sides and an outlet the other. There is an air escape vent for each half. (Figs. 1 and 2.)

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†Available from Emerson Company, Cambridge, Mass.

‡Plastic obtained from Hyolite Company, Wilmington, Del.

A drawing of the filter (Fig. 3) with the component parts labeled will best serve for description.

The filter is inserted into the arterial limb by connecting the tubing to the inlet (*C*) and the outlet (*F*). Saline is then pumped through the arterial side to rid not only the filter but the tubing of air before the blood is introduced. As the saline enters the filter it will be noticed that an air fluid level occurs just

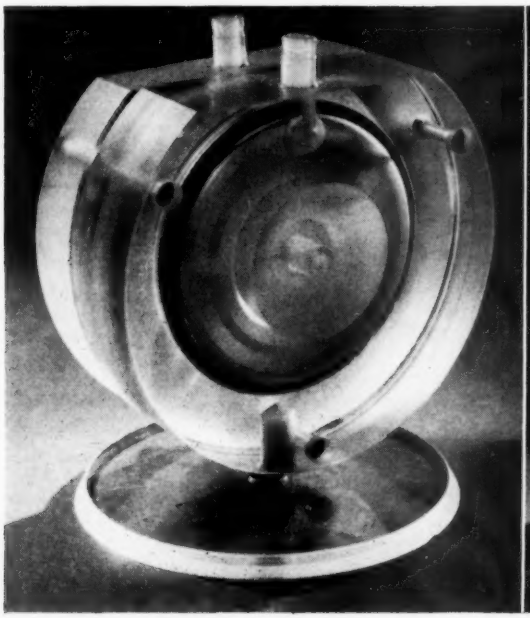

Fig. 1.—Back of filter showing inlet.

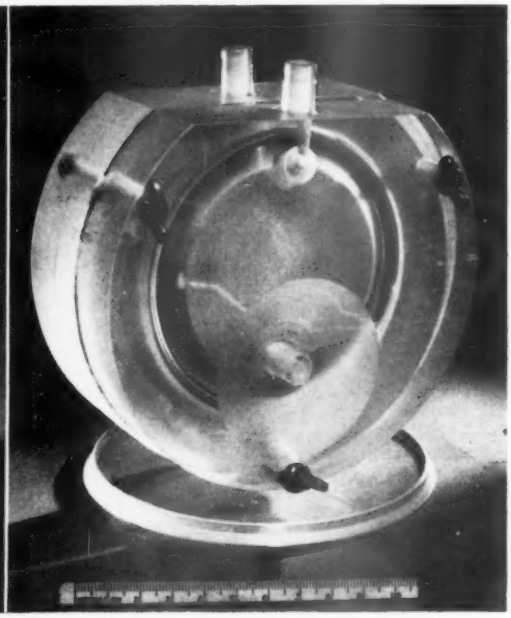

Fig. 2.—Front of filter showing outlet.

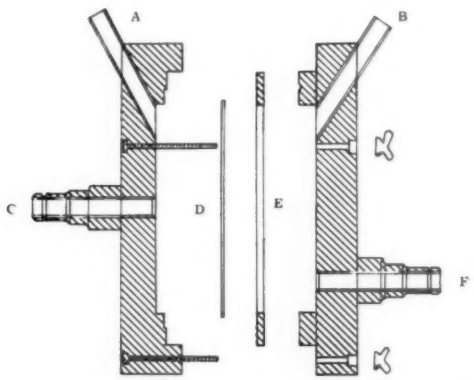

Fig. 3.—Drawing labeled to show parts: *A* and *B*, air escape vents, *C*, inlet, *D*, wire mesh, *E*, rubber gasket, and *F*, outlet. Description in text.

above the inlet level. By opening the screw clamps on the air escape vents (*A* and *B*), the air will be forced out of the rising fluid. The vents are then closed and the filter is air free. The simplicity of freeing the filter of trapped air is a great asset to its use. When the air is displaced from the filter and the tubing of the arterial side of the circuit by the saline, the oxygenator is ready for its priming blood. From the diagram it is noted that the inlet (*C*) is at

the middle of the filter body whereas the outlet (*F*) is at the bottom. The air escape vents (*A* and *B*), as the filter is circular, are at the topmost part. If any bubbles enter the filter from the reservoir during its operation, they will enter above the outlet and by the physical properties of a gas in a liquid rise to the escape vents. As the mesh is interposed between the inlet and the outlet and wet, it will not allow the bubbles to penetrate it to the outlet side during operation.

The mesh from which the filter is made is of a size that it will remove particulate matter but not blood elements. It offers no appreciable resistance to the flow of blood.

Sterilization is accomplished by opening the air vents, so that no air may be trapped, and immersing the whole in aqueous Zepherin 1: 1000 if it is made of acrylic plastic or autoclaving if made of thermal setting plastic.

After use it is cleaned by disassembling the body, discarding the mesh and gasket, and running tap water over the body. Reassembled with a new gasket and screen it is ready for sterilization and use. This entire procedure occupies only a few moments.

SUMMARY

A filter has been described, designed specifically for extracorporeal oxygenator systems. It will remove both particulate matter and bubbles from the circuit. The construction allows it to be easily used as well as readily cleaned and sterilized.

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DESCRIPTION OF A MODIFIED BUBBLE-TYPE PUMP-OXYGENATOR

Early Experimental and Clinical Experience

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BUBBLE oxygenators of simple design are enjoying more success today than was thought possible a few years ago.[4, 8] Contributing to this change has been principally a combination of three modifications over earlier designed and tested bubble or gas dispersion oxygenators.[1, 2, 6] In brief, these have been (1) nonwettable plastic material, (2) antifoam compounds, and (3) reservoirs of considerable size (1,000 to 1,500 c.c.), either helical or cylindrical, interposed between the oxygenator and patient recipient. Gaseous and fibrin emboli originating in the oxygenator, which had plagued earlier bubble-type oxygenators, have in the main been eliminated.[3, 5, 6] Increasing evidence permits one to conclude that within ill-defined time limits only minimal physiologic alterations occur during cardiopulmonary substitution with the bubble oxygenator when adequate rates of blood flow are obtained.[4, 8] Continued interest in utilizing a simple oxygenating principle for clinical pump-oxygenators has prompted re-evaluation of bubble oxygenators in this laboratory.

In the bubble oxygenator to be described, an attempt has been made to utilize certain principles concerned with the formation and elimination of bubbles from a moving liquid medium.[7] Greater understanding and utilization of these principles in the design of oxygenators will provide better equipment with low priming volume, high flow rates, minimal trauma to the blood, and increased safety. The performance of the present oxygenator has encouraged our group to further development along these lines.

THE OXYGENATOR

The oxygenator's capacity for oxygenating blood ranges from 200 to 4,000 c.c. per minute (Fig. 1). Flow is controlled by the standard unidirectional Sigmamotor finger pump with double manifolded ½ inch latex rubber tubes in the pump head. The complete priming volume of the pump-oxygenator including mixing chamber, reservoir, and tubing is at maximum 1,500 c.c.

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Contact of blood with foreign material, other than the plastic tubing used in the lines and the plastic material of the oxygenator, is limited to a few highly polished, stainless steel joints and the short latex rubber segments in the pump heads. The oxygenator is constructed of Lucite, sterilized by standard cold methods utilizing 1:1000 aqueous Zephiran.

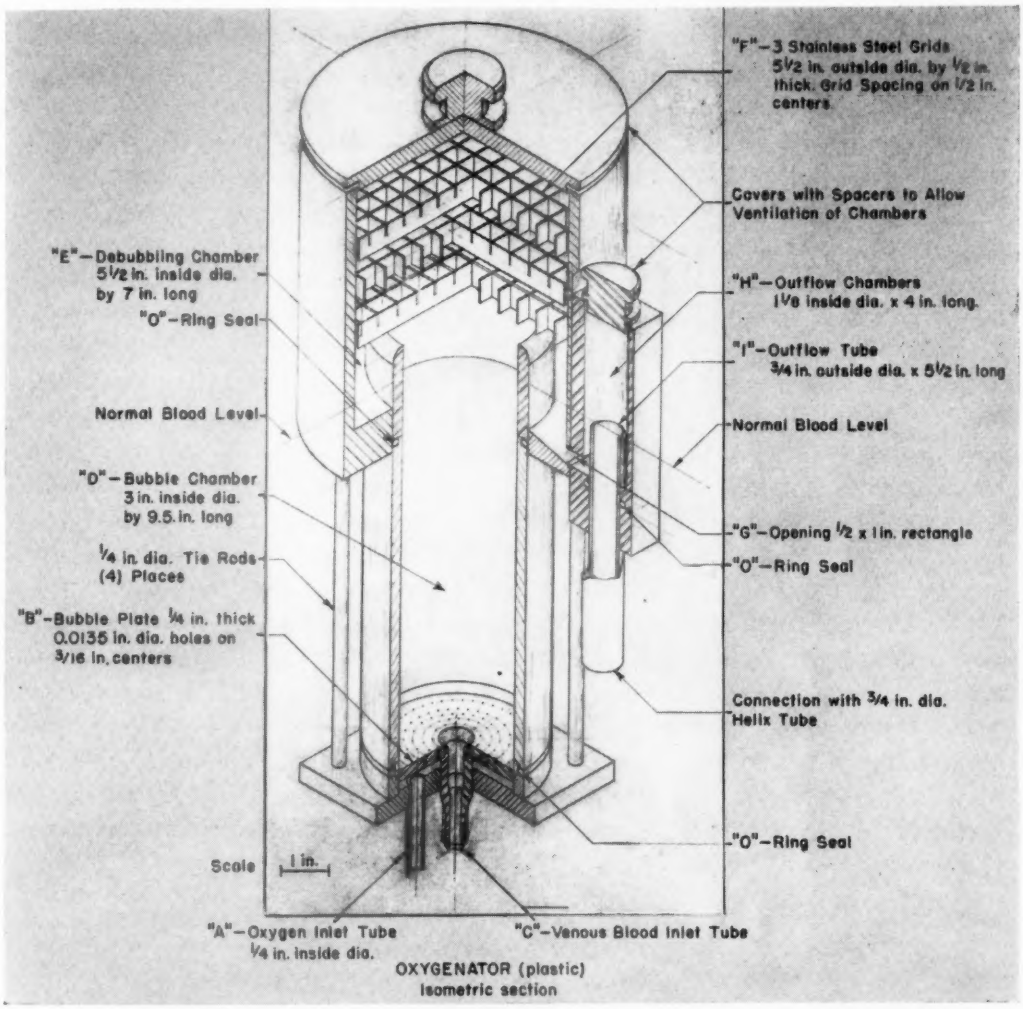
"F"—3 Stainless Steel Grids 5½ in. outside dia. by ½ in. thick. Grid Spacing on ½ in. centers.
Covers with Spacers to Allow Ventilation of Chambers
"E"—Debubbling Chamber 5½ in. inside dia. by 7 in. long
"O"—Ring Seal
"H"—Outflow Chambers 1⅛ inside dia. x 4 in. long.
"I"—Outflow Tube ¾ in. outside dia. x 5½ in. long
Normal Blood Level
Normal Blood Level
"D"—Bubble Chamber 3 in. inside dia. by 9.5 in. long
"G"—Opening ½ x 1 in. rectangle
¼ in. dia. Tie Rods (4) Places
"O"—Ring Seal
"B"—Bubble Plate ¼ in. thick 0.0135 in. dia. holes on 3/16 in. centers
Connection with ¾ in. dia. Helix Tube
"O"—Ring Seal
Scale 1 in.
"A"—Oxygen Inlet Tube ¼ in. inside dia.
"C"—Venous Blood Inlet Tube
OXYGENATOR (plastic)
Isometric section

Fig. 1.—Description of oxygenator. One hundred per cent oxygen, delivered through tube *A* at 10 L. per minute, produces a 3-mm. spherical bubble on the superior surface of plate *B*. This plate is ¼-inch thick and contains approximately 200 drilled holes (0.0135-inch diameter). Venous blood is pumped into the oxygenator through tube *C*. The mixture of venous blood and oxygen in bubble chamber *D* flows upward and overflows into the debubbling chamber *E*. Situated within this chamber and over the bubble chamber are 3 stainless steel grids (*F*); the surface of these grids is covered with Dow-Corning Antifoam A.

In operation, the bubbles disintegrate on the proximal surface of the grid. As the antifoam material loses its effectiveness, the bubbles move upward and contact fresh antifoam material on the vertical surface of the grids. The 2 distal grids serve as additional reserve units in normal operation. The bubble-free blood flows downward to the sloping bottom of the debubble chamber (*E*) and out the opening (*G*) into chamber *H*. The level of the blood in chamber *H* is determined by the level of the outflow tube (*I*). The pooled blood in chambers *E* and *H* acts as a bubble trap. Both the outflow chamber (*H*) and the debubbling chamber (*E*) are covered to reduce room air exposure. A ⅛-inch annulus permits ventilation of the chambers. The outflow tube (*I*) from the oxygenator enters the upper end of a 12-inch segment of ¾-inch I.D. Tygon tubing. This tube comprises the helical reservoir which is situated in a large Pyrex jug filled with 41° C. heated water. At the bottom of the helix, a ¾-inch to ¼-inch reducer joint connects the helix to the ¼-inch I.D. peripheral line leading to the arterial pump and thence to the patient. A filter proximal to the arterial pump is used during the initial priming of the pump and oxygenator. The filter is removed before connecting the machine to the patient.

Experimental Laboratory Work.—Twelve consecutive cardiopulmonary by-passes for 30 to 42 minutes were carried out, including right ventriculotomy incisions in 2 animals. A right thoracotomy incision was used in all animals. The right external iliac artery was used for cannulation with the arterial catheter (Nos. 16 to 18 Bardic catheters). All animals survived the by-pass and early postoperative recovery period. A 75 per cent long-term survival rate resulted with 8 animals now living 4½ to 6 months. One animal died because of an anesthetic error one hour postoperatively, and 2 animals died of bilateral hemopneumothorax with associated atelectasis during the first 24 hours. The fourth animal died 6 hours postoperatively, probably because of an incompatible blood transfusion.

All animals were perfused at flows of 45 to 50 c.c. per kilogram per minute. Under this reduced cardiac output, alterations of blood elements, O_2 and CO_2 levels, and acid-base balance have been minimal. Blood pressure levels fluctuated between 40 and 80 mm. Hg. Pre- and postoperative by-pass gas studies were obtained in these 12 experiments (Table I). When 100 per cent oxygen without additional carbon dioxide is used, a relative respiratory alkalosis occurs which has not been of any consequence during these experiments. Arterial saturation has ranged from 94.2 to 100 per cent regardless of total flow rates, the highest being 3,600 c.c. per minute. The plasma hemoglobin levels of the blood after by-pass have been elevated with a mean rise of 120 mg. per cent. This is within safe limits. Also recorded were pH ranges of these 12 by-passes (Fig. 2). Stability of this parameter is unquestionably important for the success of cardiopulmonary by-passes at reduced output and becomes more so when the overeffective ventilatory mechanism of the bubble oxygenator ceases and a large share of the acid-base balance control is returned to the lung. During this period, accumulated acid metabolites are conveniently balanced by a reduction in carbonic acid levels brought about by increased CO_2 elimination. With CO_2 levels rising following by-pass, pH reduction can be altered by the intravenous administration of sodium bicarbonate or sodium lactate, preferably the former.

TABLE I. GAS ANALYSIS AND PLASMA HEMOGLOBIN DETERMINATIONS IN TWELVE CONSECUTIVE BY-PASSES

	CO_2 VOL. %		O_2 VOL. %		O_2 SAT. %		PLASMA (HG. MG. %)	
EXPERIMENTS	PRE	POST	PRE	POST	PRE	POST	PRE	POST
12	32.1	24.8	19.3	22.6	91.4	98.3	53	173

Platelet count reduction when utilizing this pump-oxygenator is negligible (Table II). Postoperative transfusions with fresh citrated blood during protamine reversal of heparinization accounts for the essentially normal count in the recovery period. Hemorrhagic diathesis has not been a serious problem, due in part to (1) the use of fresh donor blood, (2) sterile laboratory equipment, (3) prompt replacement transfusions, and (4) slow protamine neutralization of heparin following by-pass, milligram for milligram.

These initial experiments were supplemented by additional studies which will be the subject of a separate publication.

TABLE II. PLATELET COUNTS ON FIVE BY-PASS EXPERIMENTS INCLUDING TWO RIGHT VENTRICULOTOMIES

EXPERIMENTS	PLATELET COUNT*		
	PRE-BY-PASS	30 TO 45 MIN.	1 TO 3 HOURS POST-BY-PASS
5	200,500	91,800	178,000

*In Feissley diluent (Cocaine-Na_2HPO_4 solution).

Clinical Studies.—The extracorporeal circuit used in clinical cases has been depicted diagrammatically (Fig. 3). Only in the preliminary arterialization of the priming volume is the filter depicted employed. The filter is omitted from the arterial line during actual by-pass.

Clinical experience began on Aug. 1, 1956. Eleven cases were subjected to open cardiotomy through Dec. 31, 1956. These cases are presented in Table

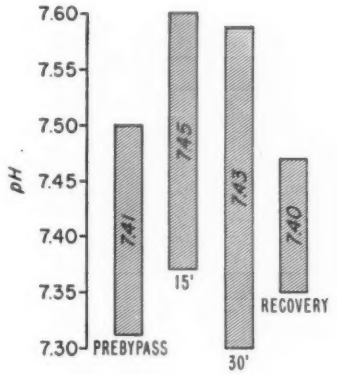

Fig. 2.

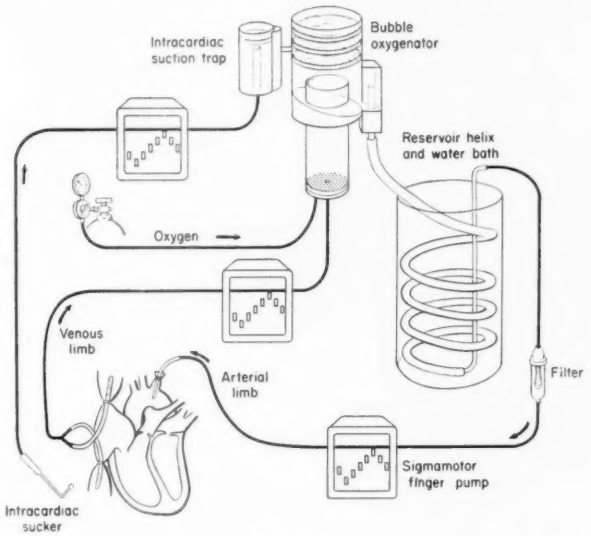

Fig. 3.

Fig. 2.—Serial pH ranges during 12 cardiac by-passes at flow rates of 45 to 50 c.c. per kilogram per minute.

TABLE III. PUMP-OXYGENATOR CASES
(Experience to Dec. 31, 1956)

PATIENT	WEIGHT (KG.)	DIAGNOSIS	FLOW (C.C./KG./MIN.)	TOTAL BY-PASS (MINUTES)	RESULTS	CARDIAC ARREST (MINUTES)
T. F.*	38	Infundibular stenosis	39	28	Survived	—
K. B.	35	Isolated pulmonic valvular stenosis	50	20	Survived	—
M. H.	50	MI, AI†	33	36	Died	—
E. F.	48	Pentalogy	30	38	Died	—
L. F.	50	MI	42	22	Survived	—
D. A.	52	IASD	44	11	Survived	—
J. W.	18	Tetrad	44	48	Died	—
P. S.	52	IASD	38	15	Survived	—
N. B.	18	IASD	55	11	Survived	—
W. M.‡	52	Tetrad	45	32	Survived	18
T. V.‡	58	IVSD	40	33	Survived	21

*Performed Aug. 1, 1956.
†Mitral insufficiency and aortic insufficiency.
‡Asystole with potassium citrate.

III. In all cases total correction has been attempted. Patients have ranged in weight from 18 to 52 Kg. with flows from 24 to 55 c.c. per kilogram per minute. Over an average by-pass time of 25 minutes, the plasma hemoglobin level has risen from an average preoperative level of 35 to 92 mg. per cent. Five patients with an average of 16 minutes of complete cardiopulmonary by-pass had less than a 50 mg. per 100 c.c. plasma hemoglobin level at the end of the procedure.

The pH of the blood prior to by-pass has averaged 7.48, with an average depression of 0.06 during the by-pass. Platelet count depression has been less than 50 per cent in 5 cases studied. No particular attempt was made to preserve platelets and standard donor bottles were used for blood collection 24 to 36 hours prior to surgery.

The mortality rate of 27 per cent is in itself meaningless, for a variety of lesions, both congenital and acquired, were subjected to surgery. Each specific type of lesion undoubtedly will eventually have a different mortality rate. Furthermore, other factors such as the degree of pulmonary hypertension, age, the degree of cardiac reserve, and so forth, will further qualify the mortality and risk in individual cases. The present series was begun as cases presented themselves and the mortality rate indicates an acceptable figure in view of the lesions treated. More important has been the behavior of the oxygenator in this clinical experience. Each death was attributable to factors other than equipment failure.

DISCUSSION

The specific aim of this oxygenator design was to produce a large number of uniform-sized bubbles (3 mm. diameter) which could readily be eliminated from the oxygenated blood. A twofold advantage is obtained by this approach, namely, small, potentially lethal bubbles are minimized and rapid gas exchange takes place due to the high $CO_2 - O_2$ pressure difference that occurs during the short contact time between the bubble and the blood. The production of bubbles of uniform size in our experience is one of the most important principles in the successful bubble-type oxygenator. Whereas the microbubbles which are produced by microholed discs, porous plates, or by trauma in eddying or turbulent streams are difficult to eliminate, the ¼-inch thick baseplate of this oxygenator, drilled with a 0.013-inch drill, produces a uniformly large-sized bubble. Therefore, these bubbles do not change in size and are satisfactorily debubbled at the undersurface of the grids coated with Antifoam.

Although the present model is constructed of Lucite and cannot be heat sterilized, an attempt has been made to evaluate heat stable plastics. Rexolite* and Epicast† appeared to have promise initially; however, both have exhibited undesirable properties and cannot be autoclaved repeatedly. More recently, stainless steel construction is being evaluated. This activity reflects our interest in obtaining a stable autoclavable unit in contrast to others interested in disposable oxygenators. The small numbers of input and output trunk lines

*Rexolite No. 1422, manufactured by Rex Corporation, West Acton, Mass.

†Epicast, Type 3, manufactured by Furane Plastics, Inc., 4516 Brazil St., Los Angeles, Calif.

needed to complete the circuit, including the ¾-inch Tygon helical reservoir, are easy to cleanse and sterilize, to a degree comparable to the care of an average surgical instrument.

The entire pump-oxygenator is mobile and easily transportable. In action the entire unit is easy to handle and only one individual is needed to operate and control the pump. The chances of technical failure or human error are reduced with a basically uncomplicated unit. This is a virtue inherent in this type of pump-oxygenator. The principles are easily understood and can be readily handled by new personnel. All components are under direct view and if for any reason technical difficulties are encountered, all parts are accessible for quick adjustment.

SUMMARY AND CONCLUSIONS

1. A simple, easily constructed bubble-type pump-oxygenator is described. A 3-mm. bubble is produced. The deliverance of a uniform-sized bubble is of importance in the success of oxygenators of the bubble type.

2. Experimentally, this oxygenator is capable of oxygenating (98 per cent saturation) venous blood at flows varying from 200 to 4,000 c.c. per minute.

3. Preliminary studies in 12 dogs yielded a 100 per cent early and a 75 per cent long-term survival rate. There is negligible trauma to the cellular elements and minimal chemical alterations of the blood.

4. The value of this type of pump-oxygenator system rests with its ease of handling, mobility, dependability, and uncomplicated operation.

5. The initial clinical experience through Dec. 31, 1956, with 11 cases utilizing this pump-oxygenator system has been summarized. There has been an acceptable degree of success without any difficulty attributable to the oxygenator.

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ADDENDUM

Since Jan. 1, 1957, 21 additional cases have been done for a variety of lesions. The original Lucite oxygenator is still being used.

EARLY TISSUE RESPONSE TO POLYVINYL SPONGE AORTIC GRAFTS

An Experimental Study

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(From the Mayo Clinic and Mayo Foundation)

WITHIN the past decade rapid development of vascular grafting techniques and increasing use of arterial grafts have stimulated widespread interest in the study of tubes fashioned from synthetic materials for arterial replacement. The ideal vascular prosthesis should be readily available and easily fashioned in various sizes and shapes to fit the individual situation. It should be pliable and elastic, should be easily sterilized, and should have sufficient strength to permit suture anastomosis, as well as strength later to withstand the incessant pounding of the arterial pulse. It should be well tolerated by the tissues and should not unduly evoke the coagulation mechanism. Autogenous and homologous arterial grafts do not fulfill all of these requirements all of the time, largely for reasons of unavailability in the proper size for the specific demand.

Polyvinyl-formal (surgical Ivalon†) sponge has been the subject of considerable investigation for arterial replacement since its tolerance by tissues and its physical properties for surgical purposes were described in 1949[3] and in more detail in 1951.[4] Studies of this material for repair and replacement of blood vessels were begun by one of us (Grindlay) in 1950. These have been reported in previous papers.[6, 7] Other favorable reports concerning the use of Ivalon in experimental arterial surgery have appeared recently,[5, 8, 9] and its successful clinical use has been mentioned.[1, 7, 8] In many respects Ivalon has appeared to meet the qualifications demanded of a synthetic arterial substitute. The present study was performed to determine in some detail the early histologic events in and about experimental abdominal aortic grafts of Ivalon.

MATERIALS AND METHOD

A segment of the abdominal aorta was replaced by an Ivalon prosthesis in 22 mongrel dogs, whose weights ranged from 5.3 to 19.5 Kg. The grafts

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†Polyvinyl-formal (surgical Ivalon) sponge used in this study was furnished by the manufacturer, Ivano, Inc., Chicago, and is obtainable from Clay Adams, Inc., New York.

varied in length and diameter, as did the aortas, but always extended from below the renal arteries to above the trifurcation (Fig. 1). The prostheses ranged from 4 to 10 cm. in length, with an average length of 7.2 cm.

Seamless tubes of polyvinyl sponge were prepared in the manner previously described.[7] Washed and frozen brick of surgical Ivalon sponge was sliced into sheets 2 mm. thick with a meat-cutting saw. These sheets were cut into strips 4 mm. wide. Uncompressed Ivalon sponge tubes were prepared with a special double-bladed borer on an electric drill press. The moistened tubes were mounted on smooth rods of brass or glass of the desired diameter and were snugly wrapped with moistened Ivalon strips. Compression to the desired degree was obtained by wrapping with 1-inch gauze under tension. The wall thickness of uncompressed sponge was 12 mm.; of sponge after compression, 1.2 to 2 mm. Fusion of the Ivalon layers, fixation in the desired shape, and sterilization were accomplished by boiling in distilled water for 20 minutes. After removal of the mold and immersion in saline solution at room temperature, the tubes were ready for use. Their inner surface appeared smooth. They were elastic and pliable.

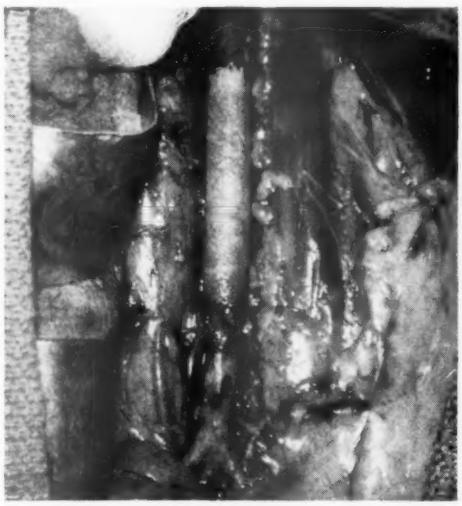

Fig. 1.—Ivalon graft at operation.

The abdominal aorta in each animal was exposed from the left with the animal under intravenous pentobarbital sodium anesthesia. All branches between the renal and iliac arteries were sacrificed. The Ivalon tube was carefully matched to the diameter of the recipient aorta and cut slightly longer than the portion of aorta to be resected to allow retraction of the aorta without undue tension at the suture lines.

Potts arterial clamps were applied to the aorta just distal to the renal arteries and proximal to the trifurcation, and the intervening segment of aorta was resected. The graft was sutured in place proximally with continuous No. 5-0 atraumatic serumproof silk lubricated with oil. Prior to performance of the distal anastomosis, the proximal and then the distal clamp were temporarily

released to allow free bleeding, and the graft and distal cuff were irrigated with saline solution to remove any collected blood. Bleeding at the suture lines following performance of the distal anastomosis and removal of the clamps was usually controlled by gentle pressure. Occasionally, an interrupted suture was necessary. Expansile pulsations were observed in all grafts, and adequate pulsations in the iliac arteries were confirmed in each instance prior to closure of the abdomen. Aortic occlusion time varied from 32 to 65 minutes. No anticoagulants were used. Frequent observation was the only special postoperative care provided.

RESULTS

Fifteen of the 22 animals had an apparently good postoperative course and were anesthetized and killed by an overdose of pentobarbital sodium at the stipulated time (Table I). Clinical signs of hind leg ischemia developed in 7 of the 22 animals. While the animal was anesthetized and prior to its death, the femoral arteries were transected. Brisk bleeding was observed in 15 animals. One of these, killed after 4 weeks, had presented no signs of hind leg ischemia while alive, yet had a thrombus completely occluding the Ivalon graft.

TABLE I. RESULTS OF POLYVINYL SPONGE AORTIC GRAFT

INTERVAL FROM OPERATION TO DEATH	NUMBER OF ANIMALS	LUMEN MAINTAINED	OCCLUSION BY THROMBUS
6 hours	2	1	1
24 hours	6	2	4
48 hours	2	2	0
72 hours	3	2	1
1 week	2	2	0
2 weeks	2	2	0
4 weeks	3	1	2
6 weeks	2	2	0
Total	22	14	8

Of the 7 animals with clinical ischemia, one showed signs of thrombosis within 6 hours after operation and 5 others within 24 hours after operation. One animal had a satisfactory course until 4 weeks after operation, when signs of ischemia developed in one hind leg. Total thrombosis of the graft was demonstrated.

The implanted prosthesis, together with immediately surrounding tissue and adjacent aorta, was removed from each animal, placed immediately, while it was unopened, in 10 per cent formalin solution, and allowed to fix for 2 or more days. Cross sections for histologic study were taken from the center of each graft and also at a distance of 1 cm. from each suture line. Two longitudinal sections, one to include each suture line, were taken. Additional sections were cut when indicated. Ivalon, after being imbedded in paraffin, is readily sectioned with an ordinary microtome without special precautions.

Gross Findings.—At the time of removal, the Ivalon graft in each animal was uniformly smooth and pliable with no apparent angulation at the suture

lines. The surrounding connective tissue was loosely adherent to the prosthetic implants until these had been in place for 6 weeks, and even then little force was required to separate the Ivalon from the surrounding tissues.

The lumen of each Ivalon implant which had remained patent was found lined by a layer of relatively dry, pale-to-deep red thrombus extending the length of the implant in each case and covering the suture lines at each end (Fig. 2). This mural thrombus varied in thickness among the animals and also from place to place in the same Ivalon tube. It was slightly eccentric at most levels in all implants and markedly so in some. Thrombosis with complete occlusion of the lumen of the graft had occurred in 8 animals. In one of these,

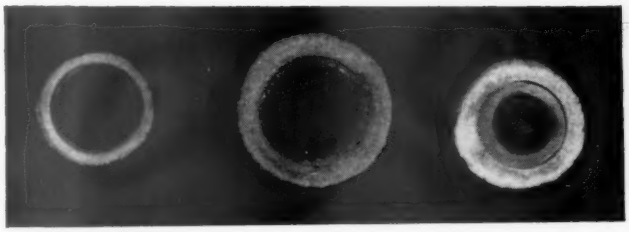

Fig. 2.—Cross sections of Ivalon grafts removed (from left to right) 3, 7, and 14 days after operation (×2).

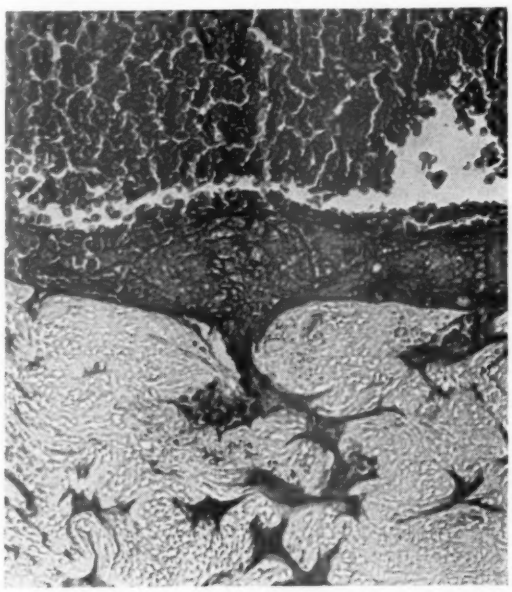

Fig. 3.—Mural thrombus 6 hours after operation. Ivalon at bottom, lumen containing blood at top. The thrombus appears to have formed first over clefts in the Ivalon (hematoxylin and eosin, ×200).

the thrombus obstructed only the distal part of the graft, including the distal suture line. In the other 7 animals, the completely occluding thrombus extended from about 0.5 cm. proximal to the proximal suture line, throughout the entire length of the Ivalon graft, and for varying distances into the distal aorta and its branches. In nearly all cases that part of the thrombus near the suture lines and within the adjacent host aorta was firmer than the deep red, sometimes gelatinous material within the lumen of the prosthesis.

Fig. 4.

Fig. 5.

Fig. 4.—Mural thrombus 24 hours after operation. Ivalon at bottom, lumen containing blood at top. Note the irregular surface of the thrombus (hematoxylin and eosin, ×100).

Fig. 5.—Highly irregular lines of Zahn in mural thrombus 24 hours after operation, suggesting turbulent flow within the graft. Ivalon at bottom, lumen at top (hematoxylin and eosin, ×200).

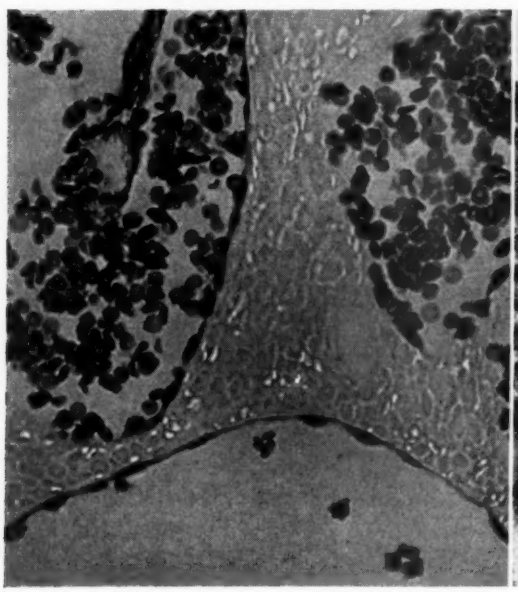

Fig. 6.

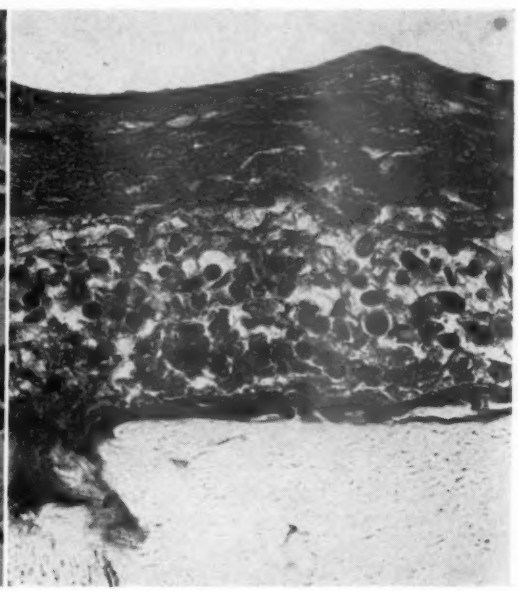

Fig. 7.

Fig. 6.—Leukocytes, including polymorphonuclear leukocytes, closely applied to the surfaces of the Ivalon trabeculae 24 hours after operation (hematoxylin and eosin, ×400).

Fig. 7.—Degeneration of leukocytes 48 hours after operation, leaving a honeycomb appearance in the thrombus. The remaining nucleated cells are predominantly macrophages. Ivalon at bottom, lumen at top (hematoxylin and eosin, ×400).

Gross inflammation in or near the site of implantation existed in the 3 animals killed 4 weeks after operation. A fibrous and purulent reaction surrounded the graft, extended up and down the aorta, and, in one animal, surrounded the kidneys, in which there was associated subacute pyelonephritis. There was a frank abscess about one of the grafts. The grafts in 2 of these 3 animals were completely occluded by thrombus, and in the third animal a thrombus at the level of the renal arteries partly occluded the aorta but the graft had remained patent.

*Histologic Findings.**—

Grafts with patent lumen: Fourteen of the 22 implanted plastic sponge prostheses retained a continuous lumen. The following histologic description, unless otherwise noted, is limited to these 14 specimens.

Within 6 hours of the time of operation, the inner surface of the Ivalon tube was lined to a depth of 10 to 500 microns by a layer of fibrin with trapped red cells, leukocytes, and aggregated platelets in its meshes (Fig. 3). Lines of Zahn were apparent only in the thicker parts of this mural thrombus, especially near the suture lines.

In those implants removed after 24 hours, the lining thrombus was thicker, varying from 50 microns to 1 mm. The inner surface was not so smooth as it seemed on gross inspection (Fig. 4), and in places irregular lines of Zahn within the thrombus suggested turbulent flow of blood in the graft (Fig. 5). Aggregates of polymorphonuclear leukocytes with lesser numbers of lymphocytes and monocytes had collected at the interface of thrombus and Ivalon. In places these leukocytes were flattened against the surface of the synthetic material, suggesting on low-power examination an endothelial-like covering of the Ivalon. This appearance was more often encountered in the interspaces of the sponge than on the intimal surface of the prosthesis (Fig. 6).

Fragmentation of some of the leukocytes was apparent in 24 hours. In those specimens removed 48 hours after implantation, leukocytic disintegration was more apparent, and there was a honeycomb appearance in the fibrin (Fig. 7). By one week after implantation, polymorphonuclear leukocytes had nearly completely disappeared from the thrombus. By this time many of the enmeshed red cells and, to a lesser extent, the platelet aggregates had disappeared, leaving in some places a thin, threadlike meshwork of fibrin (Fig. 8, *A*). Large macrophages, many containing demonstrable iron pigment, first noted in the substance of the thrombus 24 hours after placement of the graft, increased in number up to one week and then gradually diminished until at 4 weeks only an occasional such cell could be found.

One of the vessels removed after a 1-week interval had a loosely meshed mural thrombus, as illustrated in Fig. 8, *A*, throughout its entire length. Numerous intact red cells were present in one part of this loose fibrin meshwork

*We wish to express our appreciation to Dr. J. E. Edwards who reviewed the tissue sections with us and who agrees with our findings.

(Fig. 8, *B*), suggesting hemorrhage into its interstices. The lumen at this point was considerably narrowed. Similar changes were found in vessels removed after 2, 4, and 6 weeks.

A loss of lining endothelium involving 6 to 8 mm. of the host aorta adjacent to each end of the graft was apparent in all specimens removed 6 or 24 hours after operation. This denuded portion was covered in part, usually for a distance of only 3 to 4 mm., by a thin thrombus continuous with that lining the prosthetic tube. Forty-eight hours after operation partial growth of endothelium over the previously denuded part of the host aorta and beginning invasion of the overlying thrombus by fibroblasts could be demonstrated.

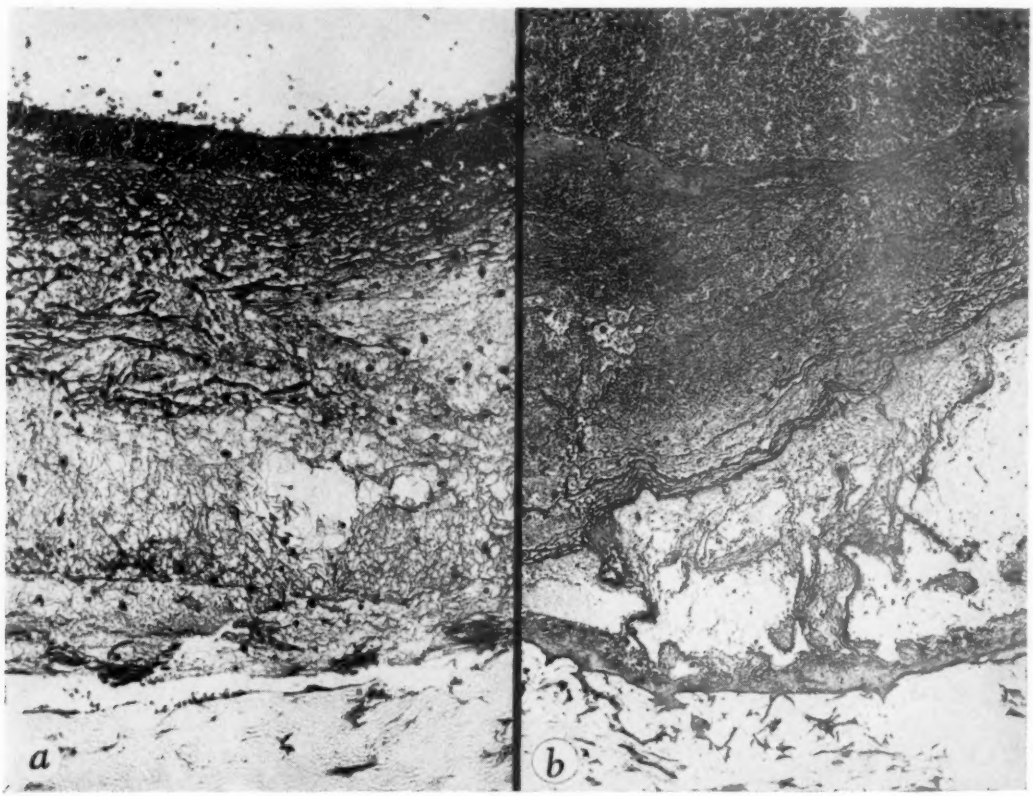

Fig. 8.—The two panels of this figure are from the same graft, removed one week after operation. *A*, The mural thrombus is composed of a threadlike meshwork of fibrin (hematoxylin and eosin, ×125). *B*, Many intact red blood cells are present in the meshwork of the thrombus, suggesting hemorrhage into its spaces. Note the cystic defects in the thrombus at bottom right. Lumen filled with blood at top, Ivalon at bottom (hematoxylin and eosin, ×60).

Seventy-two hours after implantation re-endothelization of the host aorta was complete, much of the overlying thrombus was covered by an endothelial layer, and fibroblastic invasion of this thrombus had progressed. Within one week plump-to-elongated cells resembling endothelium had overgrown the adjacent portions of thrombus within the Ivalon tube for distances up to 2 mm., and scattered fibroblasts were present within this thrombus for a shorter distance. There were many more fibroblasts within the mural thrombus adjacent to the suture line in specimens removed 2 weeks postoperatively. At this stage these

fibroblasts had extended farther into the thrombus lining the prosthesis than had endothelium covering it. Two weeks after operation that portion of the thrombus within the host aorta had been completely replaced by cellular connective tissue.

In the single specimen which had remained patent for 4 weeks, the surrounding abscess had apparently inhibited organization of the lining thrombus. Fibroblastic and endothelial proliferation in this graft were limited to the immediate vicinity of the suture line. Six weeks after operation an orderly layer of elongated cells, apparently endothelium, covered the proximal half of the lining thrombus as far as 4.5 cm. from the proximal suture line, but extended up slightly less than 1 cm. from the distal suture line. Fibroblasts within the thrombus had penetrated farther (Fig. 9).

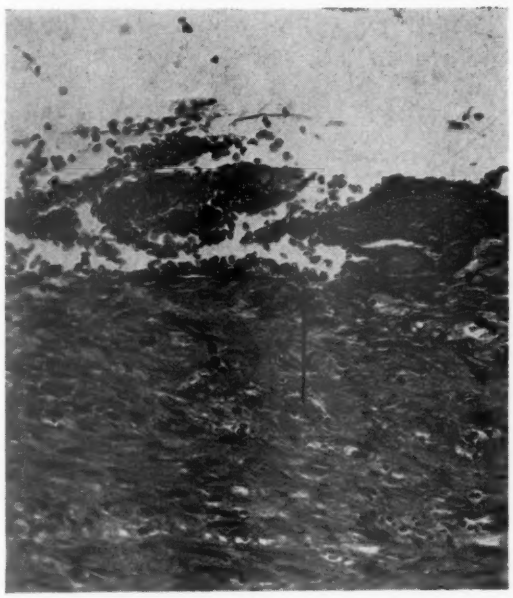

Fig. 9.—Endothelium, to left of arrow, partially covering the incompletely organized mural thrombus 6 weeks after operation. Note the irregular recent thrombus superimposed to the right of the arrow (hematoxylin and eosin, ×125).

The early inflammatory response at the graft site was similar to the reaction expected following any operative procedure involving soft tissues. The interstices of the Ivalon sponge removed 6 hours after operation were filled with serum, red cells, and scattered leukocytes, predominantly polymorphonuclear. These appeared to have entered from both the luminal and the external surfaces of the sponge and completely filled some of the spaces in the sponge 24 hours after operation. The polymorphonuclear leukocytes increased in numbers up to 72 hours after operation. By this time most of them presented evidence of disintegration. Two weeks after operation there were essentially no leukocytes in the interspaces. Polymorphonuclear leukocytes disappeared less rapidly from the adjacent connective tissue, where many were still present one week after operation. Two weeks after operation the reaction in the adventitial tissue was largely macrophagic.

Isolated macrophages, both within the outermost interspaces of the plastic sponge and in the adjacent adventitial fibrin, were first noted 72 hours after operation. They were present in greatest numbers 2 weeks after operation. Foreign-body giant cells, sometimes very large, were encountered in all specimens removed one week after operation and subsequently. These giant cells first appeared peripheral to the layer of organizing blood clot surrounding the plastic tube. They were most numerous 2 weeks after operation, when they were found within the peripheral interstices of the Ivalon and also at considerable distances adventitial to the graft. Macrophages and multinucleated giant cells in these locations were still present 6 weeks after placement of the graft (Fig. 10).

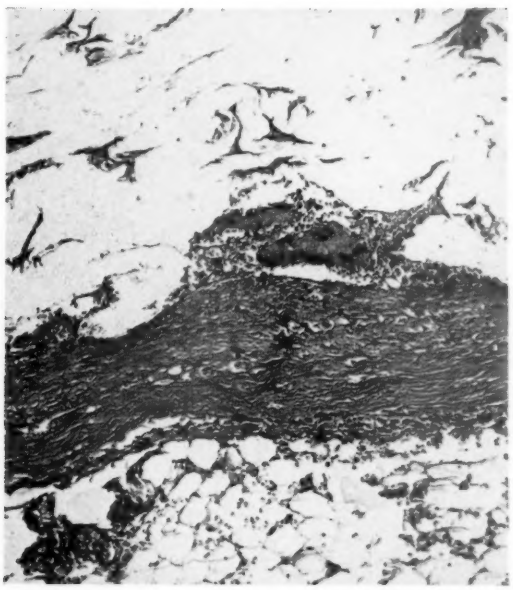

Fig. 10.—Giant cells, a few macrophages, and collagenous connective tissue surrounding an Ivalon graft 6 weeks after operation. Ivalon at top, areolar retroperitoneal tissue at bottom (hematoxylin and eosin, ×150).

A thin coagulum of fibrin and blood adjacent to the external surface of the plastic implant was apparent in those specimens removed after one week and earlier. Two weeks after operation this layer was completely organized, considerable collagen had been deposited within it, and relatively dense connective tissue surrounded the graft.

Fibroblasts and capillaries were first apparent in the outermost interspaces of the Ivalon after this had been in place for one week. The rate of ingrowth of this tissue was slow, and after 6 weeks the appearance of the graft was still largely that of an Ivalon tube encased on its adventitial surface by collagenous connective tissue and on its luminal surface by a partly organized mural thrombus. Six weeks postoperatively, connective tissue had completely penetrated the plastic sponge only near the ends of the graft. Here penetration by connective tissue from the already organized thrombus on the internal

surface apparently completed the process. Where present, connective tissue in the interspaces rapidly laid down collagen, forming dense strands within the framework of the sponge.

Grafts with complete thrombosis: In 7 of the 8 grafts occluded by thrombus the final occlusive episode had been sudden, as indicated by the coarse irregular bands of fibrin and platelets separating large pools of red cells within the lumen. In each of these 7 specimens the thrombus near the suture lines was more regularly laminated, suggesting gradual formation here. One graft, removed 4 weeks after placement, was completely filled by a concentrically laminated thrombus. In this animal there was an acute inflammatory process in the tissues about the implant and about the kidneys, as well as subacute pyelonephritis. Both the ante-mortem absence of signs of aortic obstruction and the structure of the thrombus in this animal suggested gradual occlusion over a period of time.

Acute inflammation, with gross or microscopic abscesses, involved the tissue surrounding the graft in all 3 animals killed 4 weeks after operation. Although no organisms could be found in appropriately stained tissue sections, it seemed probable that this reaction was due to bacterial contamination, especially since subacute pyelonephritis accompanied the inflammation about one graft in one animal. The specimens removed 6 weeks after operation had obviously not suffered such severe inflammation.

COMMENT

To a certain extent, each of the animals provided its own control for the study of the inflammatory response at the operative site. Until 2 weeks after operation, no great difference was detectable in the reaction immediately adjacent to the plastic sponge and that which occurred out along the recipient aorta, where this had been freed of its attachments to permit anastomosis. The changes along the aorta included the minor necrosis attendant on surgical procedures with focal acute inflammation followed by the appearance of macrophages and some foreign-body giant cells. Six weeks after operation the reaction about the host aorta had subsided with little formation of scar tissue. At the same time some macrophages and giant cells were still present about the Ivalon tube, associated with the compact collagenous connective tissue which surrounded the graft. The continued presence of macrophages and giant cells and the fibrosis were therefore attributed to the presence of the plastic. Our findings agree qualitatively with those of Bing,[2] who placed bits of Ivalon sponge subcutaneously and intraperitoneally in rats and studied the tissue response after various periods.

How much of the persisting mild reaction is due to the polyvinyl-formal sponge and how much is due to surface contaminants cannot be stated. That the sponge as available now is not completely pure (free of particulate foreign material), even after thorough washing, is suggested by the occasional presence of clear-to-brown finely granular material within the Ivalon.

The cause or causes of thrombosis, either mural or complete, in each of the Ivalon grafts in this study remain unexplained. No previous reports have stressed thrombosis as an early accompaniment of experimental arterial prostheses of Ivalon. At this writing complete occlusion has been reported in only one of 47 experimental grafts with which ours may be compared.[5, 7, 8] This single instance of thrombosis occurred in one limb of a bifurcation graft.[8] Occlusion of Ivalon grafts placed in human arteries has been related to an inadequate distal arterial bed, to flexion creases, and to suture line thrombosis.[1] Variables which need to be considered in relation to thrombosis include length and diameter of the graft, both actual and relative to the dimensions of the recipient artery, degree of compression of the Ivalon, chemical or bacterial contamination, and operative technique. We could implicate none of these factors with any constancy. The diameter of the plastic tube was carefully matched to that of the host aorta in each animal. Grafts occluded by thrombus varied in length from 6 to 9.3 cm., with an average length of 7.8 cm. Those grafts maintaining a lumen averaged 6.9 cm. long, but varied from 4 to 10 cm. in length. Previous published reports do not include sufficient detail to permit comparison.

Thickness of the wall of the Ivalon tube and size of the interspaces were not entirely constant. Within the limits of this study, no relationship of either factor to thrombosis could be demonstrated. Highly irregular lines of Zahn in the mural thrombus, interpreted as evidence of turbulent blood flow, existed in some of the specimens which had maintained a lumen. The most likely cause of turbulent flow would seem to be changes in diameter from host aorta to graft or other unevenness at the suture line however minor. Had the occlusive episodes occurred late, one would without hesitation explain them as complications of fibrinolysis with the formation of cystic spaces within the mural thrombus and subsequent hemorrhage into these spaces, as illustrated in Fig. 8. Narrowing of the lumen resulting from such a change could be demonstrated in some animals, but the 2 grafts which became nonfunctional more than 24 hours after operation had an associated acute inflammation, and their occlusion did not appear to result from such a mechanism. It was somewhat surprising to find an occluded prosthesis in one of these 2 animals. Collateral circulation by-passing the graft had been sufficient to maintain function of the hind limbs without evidence of ischemia. The occluding thrombus had apparently formed gradually by concentric additions.

SUMMARY

Polyvinyl-formal (surgical Ivalon) molded tubes were experimentally placed as abdominal aortic grafts in 22 dogs and were removed for histologic study after periods of 6 hours to 6 weeks. Within 6 hours of implantation, a thin thrombus lined the plastic prosthesis. This thrombus increased in thickness by superimposed focal or concentric thrombosis for at least 6 weeks and presented certain alterations in internal structure suggesting fibrinolysis. The mural thrombus was slowly invaded by fibroblasts from the ends of the graft

from the host aorta. Similarly, endothelium to cover the lining thrombus was found proliferating only from the host aorta from the ends of the graft toward its center.

Adventitial to the graft a thin layer composed of coagulated blood, collected polymorphonuclear leukocytes, and other phagocytes was rapidly replaced by fibroblasts, eventually leaving a dense collar of collagenous connective tissue about the Ivalon. Six weeks postoperatively, macrophages and foreign-body giant cells were still found within the connective tissue surrounding the graft.

Within the period of this study, most of the Ivalon of the graft remained uninvaded by connective tissue and was found imbedded between a dense layer of collagenous connective tissue adventitially and a partially organized mural thrombus on the luminal surface. In this study, complete permeation of the graft wall by connective tissue was found only near the suture lines, where outward growth of fibroblasts from the organizing mural thrombus could occur.

The lumen of the Ivalon prosthesis was completely occluded by a thrombus in 8 of the 22 animals. Complete thrombosis had taken place within 24 hours of the time of operation in 6 animals and at about 4 weeks in 2 additional animals. Thrombosis in these latter 2 animals was associated with acute inflammation in adjacent tissues.

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CORRECTION OF TOTAL ANOMALOUS PULMONARY VENOUS DRAINAGE

Technical Considerations

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ANOMALIES of pulmonary venous drainage are not rare, particularly when the defect involves only one lung or portion thereof. The first lesion of this type was described two centuries ago by Winslow[11] in 1739, and again later by Friedlowsky[5] in 1868. Clinical interest in this pathologic entity was stimulated more recently, however, by Brody,[1] who in 1942 published a collective review with a consideration of the types and location of such anomalies. Since that time repair of partial anomalous pulmonary venous drainage has become almost commonplace in cardiac surgery. Surgical correction of anomalous drainage of the entire pulmonary venous system has been more difficult, however, and only with the advent of techniques for open cardiac surgery has this been accomplished.

Total anomaly of pulmonary venous drainage occurs in several anatomic forms. According to Darling and associates,[4] the pulmonary veins usually enter a common venous trunk which in most instances empties into the superior vena caval system. In most cases of this type the drainage is via a persistent left superior vena cava entering the left innominate vein (Fig. 1, *a*). In a less common form of anomalous venous drainage the common trunk enters posteriorly into a normally placed right superior vena cava (Fig. 1, *b*). In another common form of total anomalous pulmonary venous drainage the common pulmonary venous chamber empties directly into the right atrium on its posterior aspect (Fig. 2, *a*). The common trunk, however, may join the coronary sinus posteriorly, which, in turn, drains into the right atrium (Fig. 2, *b*). In a rare type of total anomalous drainage, pulmonary venous blood enters the inferior vena caval system, usually via a connection to the hepatic vein. Of course, in all cases of total anomalous pulmonary drainage, regardless of anatomic type, an opening must be present in the atrial septum to support life unless the patient has some other complicated intraventricular anomaly.

Successful correction of total anomalous drainage has been accomplished recently in the type of case in which the venous connection is at the cardiac level into either the right atrium directly or via the coronary sinus (Fig. 2). Lewis and Varco[8] employed induced general body hypothermia with temporary cardiac inflow occlusion to successfully repair a case of this type. Burroughs and Kirklin[2] also reported successful results in two cases by use of

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the atrial well technique in one and temporary extracorporeal circulation in the other. We have completed the surgical repair in four patients with this type of defect during the past two years in a series of some seventy-five patients operated upon with extracorporeal circulation with a diagnosis before operation of atrial septal defect with or without anomalous pulmonary veins. In three patients the repair of the total anomaly involved enlargement of the atrial septal defect, usually with partial excision of the septum followed by transfer of the septum above the pulmonary venous orifices where it was

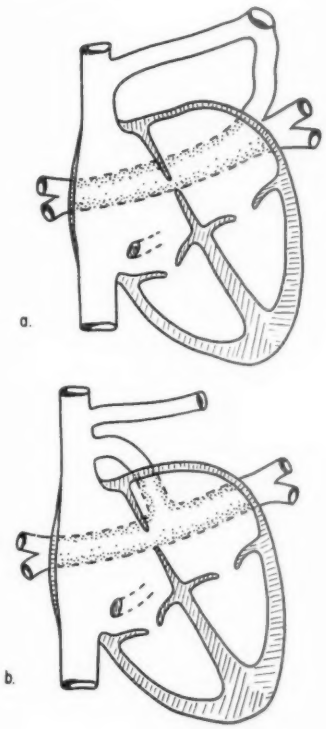

Fig. 1.

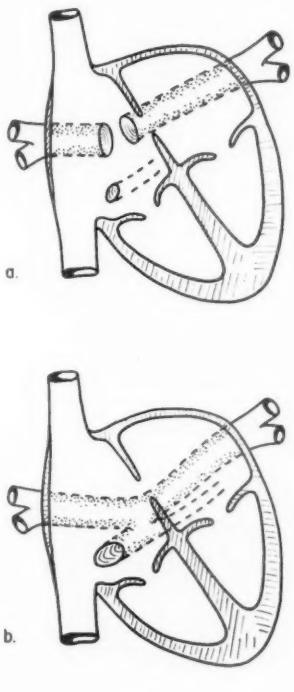

Fig. 2.

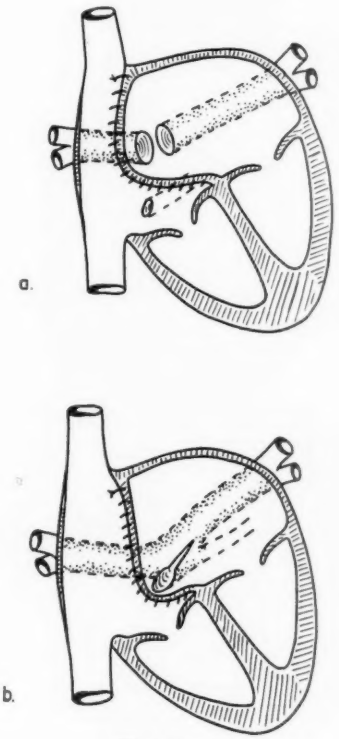

Fig. 3.

Fig. 1.—Drawings illustrating a type of total anomalous pulmonary venous drainage in which the common venous trunk enters the superior vena caval system. In *a* the trunk enters the left innominate vein via a persistent left superior vena cava. In *b* the trunk enters the posterior aspect of a normal right superior vena cava.

Fig. 2.—Drawings showing a type of total anomalous pulmonary venous drainage in which the common venous trunk enters the posterior aspect of the right atrium. In *a* the pulmonary veins enter the right atrium directly, and in *b* the veins empty into the coronary sinus which drains at the usual site of the coronary ostium.

Fig. 3.—Drawings illustrating the method of mobilization of the atrial septum with transfer of the septum to transpose the pulmonary venous orifice into the left atrium. In *a* the veins are transposed, leaving coronary sinus drainage in the right atrium (see Fig. 2, *a*). In *b* the pulmonary venous drainage, together with coronary venous drainage, is transposed into the left atrium after enlargement of the coronary sinus ostium (see Fig. 2, *b*).

sutured to the right atrial wall (Figs. 2, *a* and 3, *a*). All three of these patients survived operation. In the remaining patient in whom the pulmonary drainage entered the right atrium via the coronary sinus (Figs. 2, *b* and 3, *b*), the coronary sinus ostium was enlarged and displaced into the left atrium when the atrial septum was transposed. This patient also survived operation and no visible cyanosis resulted from coronary venous blood entering the left side of the heart. Two other patients with this anomaly were encountered

who did not survive operation. In one cyanotic patient, aged 42 years, in severe congestive heart failure, ventricular fibrillation and death ensued shortly after the thoracotomy incision. The other patient, a 5-year-old girl, died seven days after operation, at which time an anomalous drainage of the inferior vena cava into the azygous vein was discovered. The hepatic vein was draining normally into the right atrium. Cyanosis was evident after operation and at autopsy it was discovered that the splenic vein emptied independently into the left atrium.

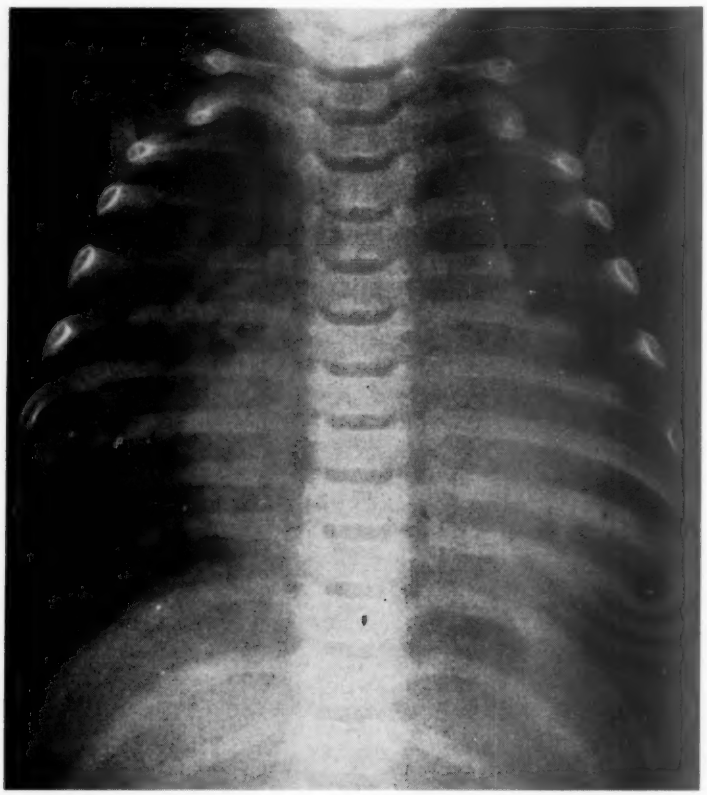

Fig. 4.—Roentgenogram of chest in 6-month-old infant showing generalized cardiac enlargement and increased pulmonary vascularity.

Apparently, from our perusal of the surgical literature, all previous attempts at complete correction of total anomalous pulmonary venous drainage into the superior vena caval system (Fig. 1) have been unsuccessful although a number of patients survived partial correction. Partial correction of the anomaly has usually included anastomosis of the left superior vena cava to the left atrium without closure of the atrial septal defect, or incomplete occlusion of the remnant of the left superior vena cava beyond the anastomosis.[6, 9, 10] Burroughs and Kirklin[2] reported a 6-month-old infant in whom correction was performed using a pump oxygenator. In this case the left superior vena cava was ligated and divided, and the common venous trunk was anastomosed side-to-side to the left atrium from the left side of the mediastinum. The atrial septal defect or patent foramen ovale was repaired through a right

atriotomy. The patient died eight hours after operation in pulmonary edema, and at autopsy the left side of the heart was discovered to be underdeveloped. Others have commented upon the small size of the left atrium in this anomaly, and Keith and associates[7] stated that the "left auricle is underdeveloped and thus cannot be expected to carry the total blood flow adequately." Another disadvantage in the method of anastomosis of the common venous trunk to the left atrium in reported cases is the fact that the atrium and appendage when anastomosed to the vein may predispose to kinking; also the size of the opening may be compromised by the small size of the cardiac chamber.[4, 10]

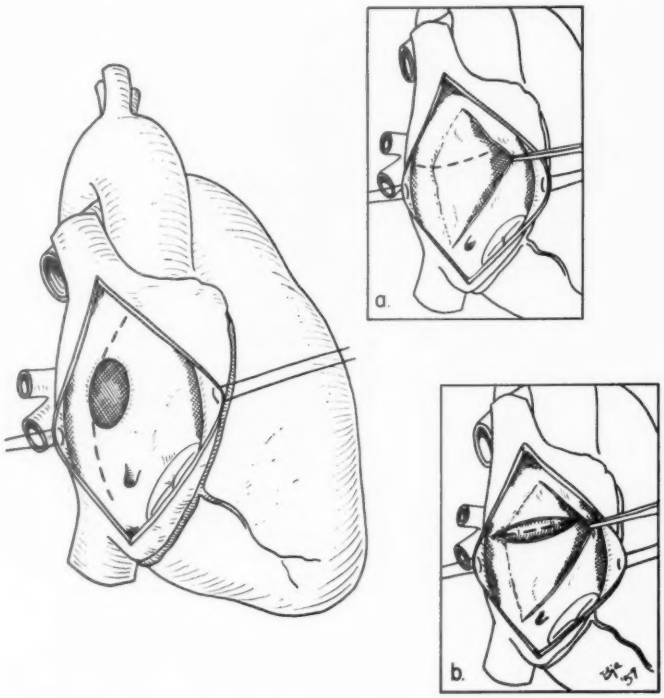

Fig. 5.—Drawings showing method of repair of total anomaly of pulmonary veins using temporary cardiopulmonary bypass. The right atrium was incised to expose the atrial septum and foramen ovale defect. Position of septal incision is indicated. After exposure of the posterior aspect of the left atrium by retracting the edge of the septum to the left, a long incision was made in the wall of left and right atrium (*a*). In *b* is shown position of incision in common venous trunk as viewed through the posterior atriotomy incision.

Furthermore, it is usually necessary to displace the dilated heart during the performance of the anastomosis under awkward conditions. Suffice it to say that our own experience with these cases done by such methods was unsatisfactory, and none survived until the development of a new technique. Recently we have been successful through use of temporary cardiopulmonary bypass in completely correcting the anomalies in a case illustrated by Fig. 1, *a*, and since this appears to be the first instance in which this has been successfully accomplished, we believe that a description of the surgical technique employed is worth while.

CASE REPORT

M. L. J., a 6-month-old white male, was admitted to the Texas Children's Hospital, Houston, Texas, on July 16, 1957. Transient cyanosis on crying was noted soon after

birth, and cardiac enlargement and a murmur were noted subsequently. Recurrent respiratory infection developed, and digitalization was carried out with slight improvement.

On admission the patient was a weak, 10 pound infant who was cyanotic on crying. The heart was enlarged and a soft systolic murmur was noted along the left sternal border with an apical gallop rhythm. The lungs were free of râles and the liver was palpable 2 cm. below the costal margin.

Laboratory studies revealed a normal hemogram with hemoglobin 13.1 Gm. per cent and normal urinalysis. Electrocardiogram showed a sinus rhythm and suggested right atrial and right ventricular hypertrophy. Roentgenograms of the chest revealed generalized cardiac enlargement and increased pulmonary vascularity (Fig. 4).

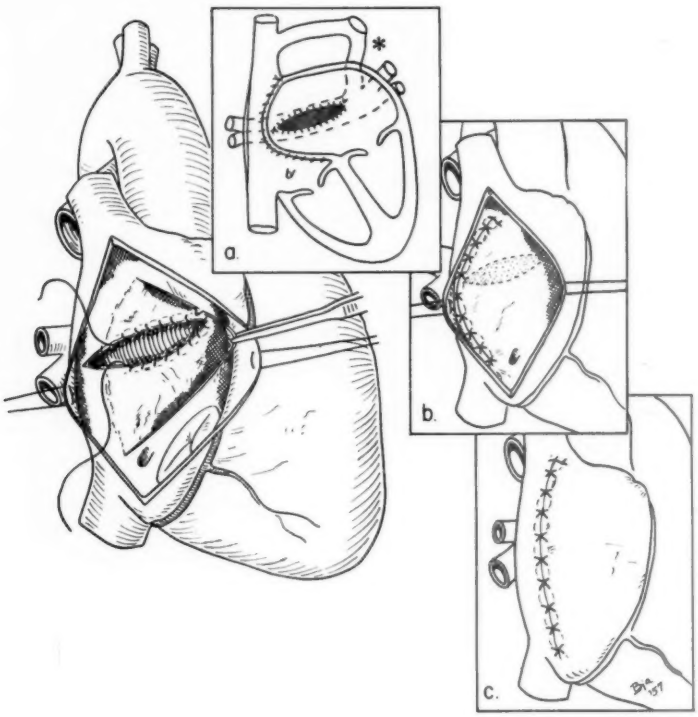

Fig. 6.—Drawings showing method of creating maximum size anastomosis between common venous trunk and posterior aspect of left and right atria. In *a* the plan of operation is shown, including the anastomosis and the left atrium enlarged by repositioning of the atrial septum. Ligation of the left superior vena cava (asterisk) completes the correction of the anomaly. In *b* and *c* are shown the repair of the atrial septal defect and right atriotomy incisions, respectively.

Cardiac catheterization* under trichlorethylene anesthesia revealed the following essential findings:

	Pressure (mm. Hg)	*O_2 Sat. (%)*
Superior vena cava		85
Left innominate vein		95
Inferior vena cava		66
Right atrium	25/5	78
Right ventricle	115/0	78
Pulmonary artery	115/70	78
Femoral artery	75/50	84

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Angiocardiography revealed an interatrial communication and the late appearance of contrast medium in a persistent left superior vena cava, confirming the diagnosis of total anomalous pulmonary venous drainage.

Operation was performed on July 31, 1957, under general anesthesia through a bilateral anterior thoracotomy incision entering the right fourth intercostal space and the left third space transecting the sternum. The pericardium was incised, and mediastinal exploration revealed the left superior vena cava containing highly oxygenated pulmonary venous blood which was emptying into the left innominate vein.

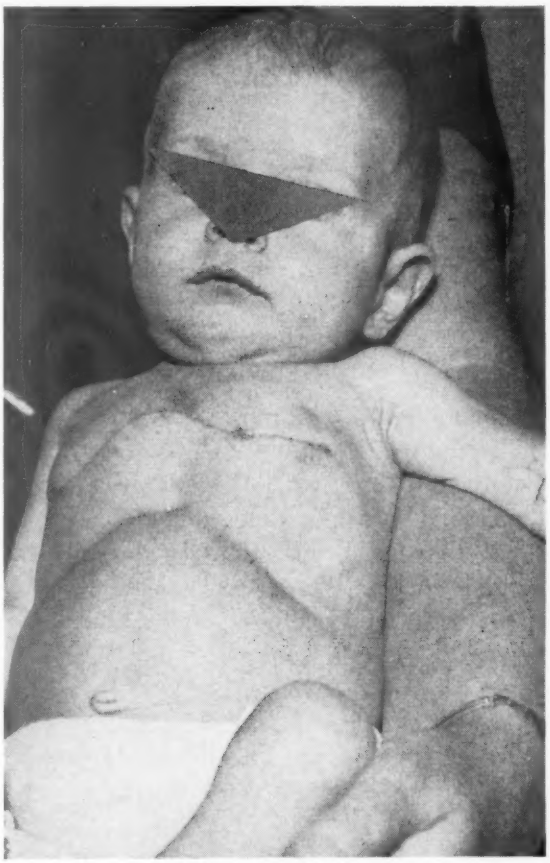

Fig. 7.—Photograph of patient made thirteen days after operation before dismissal from hospital.

Without further dissection, preparations were made for cardiopulmonary bypass using a pump oxygenator system previously described.[3] The superior and inferior venae cavae were cannulated through the right atrium for venous outflow, and the right common femoral artery was cannulated for return of oxygenated blood. After heparinization the total body perfusion and cardiopulmonary bypass was started at a rate of 50 c.c. of blood per kilogram body weight per minute (220 c.c. per minute total). The left superior vena cava was occluded with a tape tourniquet.

The right atrium was incised parallel to the direction of the venae cavae, and the foramen ovale defect measuring 15 mm. in diameter was identified (Fig. 5). With this defect as a starting point, the atrial septum was detached from its right lateral junction with the atrial wall thus gaining wide access to the left atrium (Fig. 5, *a*). The mitral valve was small but appeared to be adequately developed. No pulmonary venous ostia were present in the left atrium and the atrium was underdeveloped.

A transverse incision was made through the posterior wall of the left atrium in a line parallel to the common venous trunk, with the incision extending upward on the right to include part of the right atrial wall (Fig. 5, *a*). A similar incision was made 2.5 cm. long in the pulmonary venous trunk (Fig. 5, *b*). Using 5-0 arterial silk sutures, the venous trunk was anastomosed to the posterior wall of the left atrium, making the anastomosis through the right atriotomy incision without displacing the heart (Fig. 6). A large anastomosis was created, incorporating part of the right lateral atrial wall. Upon completion of the anastomosis the atrial septum was sutured to the right atrial wall above the anastomosis so that the resulting left atrium was considerably enlarged (Fig. 6, *a* and *b*). Finally, the right atriotomy incision was closed with continuous silk sutures (Fig. 6, *c*), and the tourniquets occluding the superior and inferior venae cavae were released.

Cardiac action continued throughout the twenty-three-minute period of cardiopulmonary bypass. The femoral arterial pressure remained low for several minutes until the left superior vena cava was doubly ligated, directing the entire pulmonary venous flow into the left atrium (Fig. 6, *a*). The patient's condition improved steadily thereafter. Upon completion of the operation, a tracheostomy was performed.

During the postoperative period recovery was satisfactory except for a period of transient cyanosis caused by atelectasis of the left upper lobe on the day following operation. Tracheostomy facilitated the management of this complication. The patient was discharged from the hospital on the thirteenth day after operation in excellent condition without signs of congestive failure (Fig. 7).

DISCUSSION

In the past, surgical efforts to correct the most common type of total anomaly of pulmonary venous drainage have almost uniformly failed because of the critical condition of the patients, the complicated nature of the anomaly, and the obvious need for open cardiac conditions for complete repair. In our own early experience with several patients who had total drainage through a persistent left superior vena cava, cardiac standstill tended to occur when the anomalous vessel was partially occluded for anastomosis to the left auricular appendage. Even slight displacement of the dilated heart to expose the venous trunk and diminutive left atrium from the left side of the mediastinum also produced severe bradycardia, preventing in most instances satisfactory exposure. In instances where an anastomosis was accomplished, the opening was either too small to accommodate total venous flow, or the left atrium was too underdeveloped. In any event, all of these attempts were unsuccessful.

The need for open cardiac conditions was evident in order to repair the atrial defect at the same time the venous anomaly was corrected. Conceivably this could be accomplished under hypothermia, but the limitations on time available for intracardiac operations by the hypothermic technique would appear to make this method less satisfactory than cardiopulmonary bypass. In our opinion, the requirements for safety and completeness in correction of this complicated anomaly are fourfold. First, cardiac and pulmonary function should be provided mechanically by a pump oxygenator in these patients who are frequently in congestive heart failure. Second, the largest possible anastomosis must be created between the common venous trunk and the left atrium without significant displacement of the heart during performance of the anastomosis. A side-to-side anastomosis appears to be more satisfactory than end-to-side and prevents subsequent torsion and possible kinking of veins. Third, the septal defect must be closed and the underdeveloped left

atrium must be enlarged to accommodate the pulmonary venous return without obstruction. Fourth, the persistent left superior vena cava or anomalous connection to a right superior vena cava must be ligated. All of these criteria appear to be fulfilled by the technique employed on the patient reported herein.

SUMMARY

In the most common type of total anomalous pulmonary venous drainage, the venous flow is directed into a persistent left superior vena cava which empties into the left innominate vein. In the past, successful complete correction of this defect has not been accomplished for a number of reasons. Requirements for correction of this defect include (1) use of temporary cardiopulmonary bypass, (2) creation of an ample anastomosis between the common pulmonary venous trunk and left atrium, (3) closure of the atrial septal defect and enlargement of the left atrium, and (4) ligation of the anomalous pulmonary venous channel joining the superior vena caval system.

A case is reported in which a 6-month-old infant was successfully operated upon, and a description is made of the technique employed which fulfills the criteria for a satisfactory operation.

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THE RECOGNITION AND TREATMENT OF PNEUMOTHORAX ACCOMPANYING RADICAL NECK DISSECTION

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THE consequence of an obstructed airway in association with surgical exposure of the superior mediastinum† may be the development of mediastinal emphysema and bilateral pneumothorax. As a result, both of these complications not infrequently accompany operations about the neck. Their clinical manifestations, however, as well as the urgency of recognition and adequate treatment have received relatively little emphasis. In the older literature, these complications were better appreciated as hazards of tracheostomy in infants and partial thyroidectomy.[3, 7, 12] With modern anesthetic techniques, particularly the employment of endotracheal anesthesia, these problems have received less attention and are either overlooked in discussion on more extensive operative procedures about the neck or are mentioned in a rather casual manner.[4, 6, 8, 11, 16] The development of pneumomediastinum or pneumothorax in some degree is probably more frequent than is realized since with minimal to moderate involvement there may be no obvious clinical manifestations. In those individuals in whom these complications have been recognized and reported, the morbidity and mortality have been appallingly high for a condition that occurs during surgery and for one that can be so simply and effectively managed once the diagnosis has been established. The same principles of treatment that have been advocated in the management of pneumothorax accompanying traumatic wounds of the chest are just as applicable in this circumstance. The increasing frequency of radical neck dissections demands that everyone undertaking this procedure be cognizant not only of the incidence and mechanism of pneumothorax but, equally important, the ease of recognition and the urgency of immediate and adequate treatment.

Champneys[7] in 1882 was the first to emphasize the role of pneumothorax as a cause of death following operations about the neck, particularly tracheostomy in infants. He arrived at this conclusion while studying the efficacy of various methods of artificial respiration. By means of a tracheal cannula connected to a water manometer in stillborn children, the ventilatory exchange could be measured. In the course of these observations, Champneys noted that pneumomediastinum occurred in 7 of 21 experiments, and in 5 of these

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†It should be recalled that the superior mediastinum extends well into the neck since it is that portion of the mediastinum above the level of the pericardium that is bounded behind by the bodies of the upper 4 thoracic vertebrae in front by the manubrium and the 2 straplike depressor muscles of the larynx. (From Grant, J. C. B.: Method of Anatomy, Ed. 5, Baltimore, 1952, Williams & Wilkins Company.)

there was associated pneumothorax. He correctly concluded that "The conditions favoring its (mediastinal emphysema) production are two: a wound of the deep cervical fascia and obstruction to the air passages; the moment of its production is inspiration." On this basis he was able to predict that pneumothorax would be found to be a common cause of death following tracheostomy in infants. Since that time there have been several reports citing examples following other operative procedures about the neck.[1, 3, 9, 10, 12, 14, 16] Because of the type of reference (individual reports) as well as that in many instances the diagnosis was made only at autopsy, it is difficult to appraise either the incidence or morbidity of mediastinal emphysema and pneumothorax.

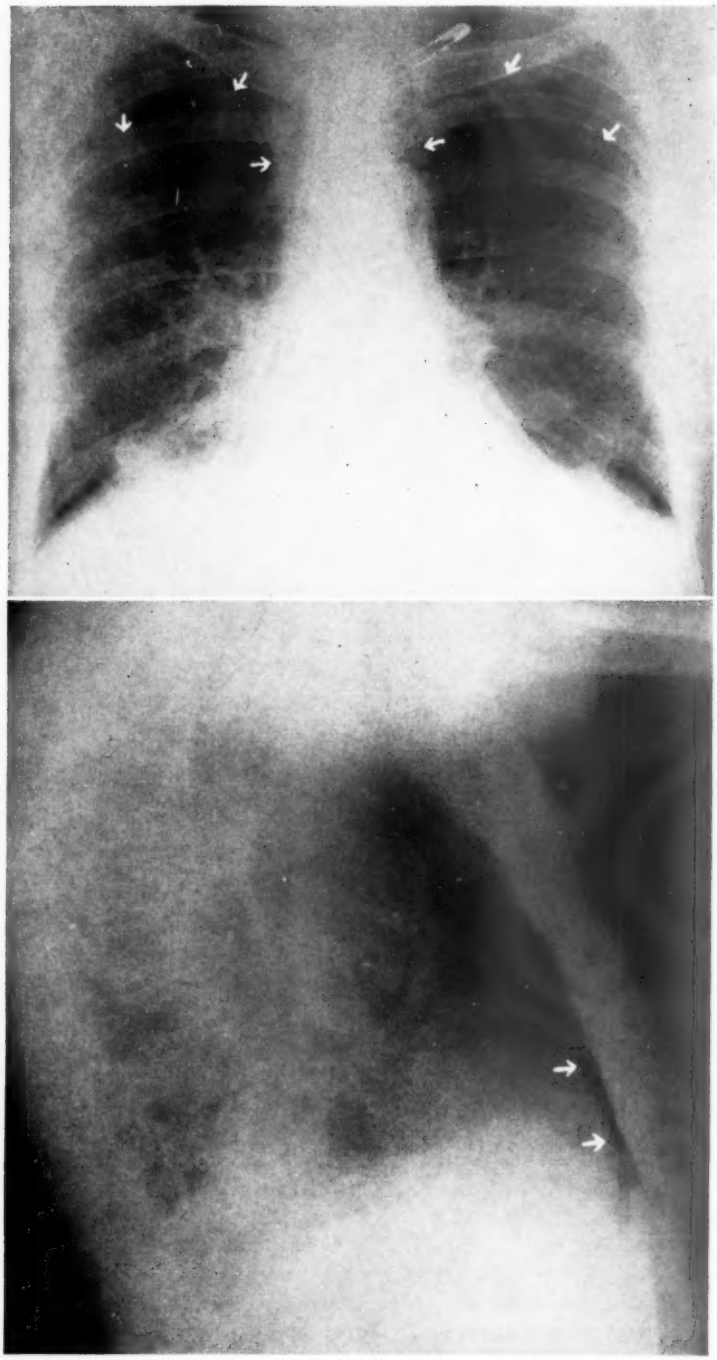
In 1950 Bowden and Schweizer[5] cited their experience with 18 cases; however, the relative incidence was not included. In this group, there was a 28 per cent mortality but in only 2 patients could death be attributed directly to pneumothorax. Most disconcerting was a report by Aiken and Smith[2] in 1952 presenting 8 patients with a 50 per cent mortality. This mortality rate may be related to the method of diagnosis (since confirmation of a suspected pneumothorax was usually by way of chest roentgenogram) and inadequate therapy. In our experience pneumomediastinum and pneumothorax have occurred 4 times in the past 4 years in 37 patients experiencing radical neck dissections. Such an unusually high incidence may be related to the type of operative procedure as well as awareness of this complication. The course and management of these patients seemed sufficiently instructive to report them in detail.

CASE REPORTS

CASE 1.—W. T. S., a 50-year-old white farmer, was admitted to North Carolina Memorial Hospital on July 2, 1956, 2 months following irradiation of a squamous cell carcinoma of the tongue. Examination disclosed a man in good nutritional status and in apparent good health. An x-ray of the chest was within normal limits. Under endotracheal anesthesia, employing a semiclosed system and utilizing sodium Pentothal and succinylcholine for induction and ether, cyclopropane, and nitrous oxide for maintenance, a left radical neck dissection was carried out. Respirations were, for the most part, spontaneous and at no time was there abnormality in the blood pressure, pulse, or respiration. During the latter part of the operative procedure because of a sucking sound noted in the superior mediastinum during inspiration, a diagnostic thoracentesis was advised. This was accomplished through the left second interspace employing a No. 15 needle. Air was obtained; however, the surgeon interpreted this as artifact due to a faulty syringe. Even though second aspiration disclosed air, this was also interpreted as artifact. On the day following surgery, a roentgenogram of the chest disclosed widening of the superior mediastinum with a moderate pneumomediastinum as well as a moderate right and left pneumothorax (Fig. 1). There was no evidence of progression on subsequent roentgenograms and the pneumothorax and pneumomediastinum cleared spontaneously. The patient's postoperative course was uncomplicated, and he was discharged on the tenth postoperative day.

Comment: This patient must have developed mild respiratory tract obstruction sometime during his operative procedure with a resulting superior mediastinal emphysema. Although this resulted in a bilateral pneumothorax, it did not cause sufficient change in pulmonary ventilation to be manifest. The patient recovered satisfactorily. However, once the diagnosis was established, bilateral intercostal tubes should have been inserted. Without a postoperative chest x-ray, this complication might well have been overlooked since at no time did there develop evidence of subcutaneous emphysema.

A.



B.

Fig. 1.—Roentgenograms of the first patient on the second postoperative day disclosing mediastinal emphysema as well as bilateral pneumothorax with partial collapse of each lung. The presence of air in the mediastinum as well as both pleural cavities is evidence of the mechanism of development. Arrows indicate extent of pneumothorax and pneumomediastinum.

CASE 2.—B. H., a 61-year-old white man, was admitted because of a moderately well-differentiated epidermoid carcinoma of the epiglottis with involvement of regional lymph nodes. The patient's health was otherwise excellent and the remainder of the physical examination was within normal limits except for mild emphysema. Treatment consisted of a total laryngectomy and a right radical neck dissection carried out under endotracheal anesthesia employing a preliminary tracheostomy. Anesthesia was maintained through a semiclosed system utilizing oxygen, nitrous oxide, and intravenous meperidine hydrochloride. Respirations at the beginning of the operation were controlled but later assisted. During the operative procedure, there was moderate bronchial secretion requiring occasional aspiration. At the termination of the operative procedure, a permanent tracheostomy was established and the anesthesia discontinued. Respirations at this time were quite labored and only slightly improved by tracheal aspiration. The blood pressure fell to 80/40 mm. Hg with an associated tachycardia of 120. Estimated blood loss had been replaced. The respirations became even more labored, diaphragmatic in character, and with a spontaneous rate of 11 per minute. Intratracheal oxygen only partially relieved the patient's cyanosis. Auscultation of the chest disclosed respiratory wheezes as well as flapping sounds in the lower chest. A diagnostic thoracentesis disclosed air to be present bilaterally. Bilateral anterior chest tubes were inserted in the second intercostal space and connected to water-seal drainage. Within 10 to 15 minutes following the evacuation of considerable air by way of the chest tubes, the blood pressure returned to normal, pulse slowed, respirations approached normal, and cyanosis cleared. Chest x-rays on the day following surgery disclosed the lung to be fully expanded with minimal atelectasis. The intercostal tubes were removed on the first postoperative day, and the patient's subsequent course was uneventful.

CASE 3.—D. F., a 29-year-old white woman, was seen because of carcinoma arising in the left lobe of the thyroid with metastases to lymph nodes on the left side of the neck. Previous health had been excellent and physical examination was otherwise within normal limits. Chest x-ray failed to disclose any abnormalities. Under endotracheal anesthesia employing sodium Pentothal, nitrous oxide, and cyclopropane, a total thyroidectomy and left radical neck dissection were performed. During the operation there was moderate dissection in the superior mediastinum necessitated in ligating the stump of the internal jugular vein. Shortly, thereafter, resistance in ventilation of the patient was noted and a diagnosis of pneumothorax entertained. Diagnostic thoracentesis disclosed air to be present bilaterally and bilateral anterior thoracotomy tubes were inserted at the second intercostal space and connected to underwater drainage. This complication occurred shortly after the onset of the neck dissection, and with ease of ventilating the patient the operative procedure was continued without further incident. Postoperative x-rays disclosed a residual pneumothorax to be present on the right. On the second postoperative day, both lungs were fully expanded and the chest tubes were removed. The subsequent course was uneventful.

CASE 4.—T. C., a 44-year-old white woman, was admitted because of carcinoma of the thyroid arising in the right lobe being associated with metastases to lymph nodes of the neck. The patient's health was otherwise excellent and on physical examination, the pertinent findings consisted of an enlarged lobe of the thyroid on the right with a palpable mass in the right anterior cervical triangle. The remainder of the examination was within normal limits. Under endotracheal anesthesia employing a semiclosed system with cyclopropane and ether, a total thyroidectomy and right radical neck dissection were performed. The procedure was well tolerated by the patient. However, following extubation, there was considerable respiratory distress associated with a drop in blood pressure and pulse. The respirations became more labored with evidence of suprasternal and intercostal retraction. Immobile vocal cords suggested laryngeal obstruction; however, there was no relief of the respiratory difficulty with reintubation. The diagnosis of pneumothorax was confirmed by bilateral diagnostic thoracentesis through the second anterior intercostal

space. Bilateral anterior chest tubes were inserted through the second intercostal spaces and a tracheostomy was performed. The patient's response to this therapy was excellent. On the second postoperative day there was no residual pneumothorax by x-ray and the chest tubes were removed. The remainder of the postoperative course was uncomplicated except for a transient vocal cord paralysis. Recovery of vocal cord function was sufficient at 3 months to permit closure of the tracheostomy.

Comment: This patient's recurrent nerve injury with resulting vocal cord paralysis and laryngeal obstruction undoubtedly aggravated her respiratory difficulties and either initiated or enhanced the pneumothorax.

DISCUSSION

These 4 patients well illustrate the mechanism and manifestations of pneumomediastinum and pneumothorax accompanying operations about the neck. In none could the pneumothorax be attributed to the accidental opening of the pleura. In all instances the superior mediastinum was exposed and there was probable respiratory tract obstruction with an increased inspiratory effort.

Unexplained respiratory distress or deterioration of cardiovascular dynamics in association with mild respiratory difficulties occurring during operations about the neck and particularly when the superior mediastinum or trachea has been exposed should immediately arouse suspicion of pneumothorax. Any of the consequences of hypoxia, namely, cyanosis, tachycardia, cardiac arrhythmia, or hypotension, may be the chief manifestation in the patient. Disparity between the movements of the breathing bag and the respiratory effort of the patient may also be indicative.[17] Thus, in the second patient, cyanosis, tachycardia, and a falling blood pressure unassociated with blood loss led to the diagnosis. The third patient exhibited only difficulty in controlling ventilation without change in cardiovascular dynamics and no detectable respiratory tract obstruction. The fourth individual had labored respiration following extubation with associated fall in blood pressure and bradycardia. This patient was not improved following reintubation suggesting some other cause for inadequate ventilation. Only in the first patient was a sucking sound noted in the superior mediastinum, and on this basis the diagnosis entertained.

Once the diagnosis has been considered, immediate confirmation by needle aspiration of *both* pleural cavities should be performed. Although occasionally air may be heard bubbling in the superior mediastinum during respiratory movements, it is uncommon to be able to recognize the ingress of air through the superior thoracic inlet, and this cannot be used as a reliable method of diagnosis. The physical findings of resonance on percussion, diminished breath sounds, or crunching sounds on auscultation are helpful but too often inconclusive. Confirmation of a suspected pneumothorax by radiologic examination of the chest during surgery is to be condemned. Such an examination is not only time consuming, but the delay in therapy may be extremely hazardous. Furthermore, an early pneumothorax may be readily overlooked. Once pneumothorax has occurred, even though the patient is intubated and particularly if respiration is voluntary, the greater the respiratory effort, the greater will be the associated pneumothorax. Regardless of the extent of the

surgical procedure in the neck, a diagnostic thoracentesis should always be performed *bilaterally*. Diagnostic aspiration of intrapleural air is readily accomplished through the second or third intercostal space anteriorly. This area of the chest has usually been prepared and should be included in the sterile field. Once air has been demonstrated, bilateral anterior thoracostomy tubes should be inserted. Needle aspiration of the pleural cavity is inadequate and will be accompanied by higher morbidity and mortality. Usually with bilateral catheter drainage of the pleura and adequate ventilation, there is rapid recovery and the operative procedure may be continued. The thoracostomy tube may be removed on the first postoperative day once it has been demonstrated by roentgenograms that the lung has re-expanded.

Subcutaneous emphysema has been reported in such patients but was not observed in our experience. This should be regarded as a fairly late but inferential finding. It is most likely to appear in those individuals who have not had sufficient compromise of pulmonary ventilation to suggest the possibility of pneumomediastinum or pneumothorax.

The mechanism of pneumothorax occurring during operation about the neck has been well described[5, 7] but is still not generally appreciated.[15] It is not incident to pleural injury occurring during the course of the dissection. Sibson's fascia forms a heavy protective investment over the lung and is not readily penetrated. Furthermore, such a mechanism would not explain the pneumomediastinum that precedes the pneumothorax nor the bilaterality of the pneumothorax. The superior mediastinum communicates with the structures of the anterior neck by way of a potential space formed by the pretracheal fascia compressing and joining the carotid sheath and deep cervical fascia, which again joins the carotid sheath behind the trachea. As has recently been pointed out, there are no well-defined fascial planes surrounding the trachea and great vessels of the neck.[13] These areas are invested in areolar tissue. Mere opening of the superior mediastinum as accompanies exposure of the trachea or that required for radical neck dissections with ligation of the internal jugular vein gives sufficient access for development of mediastinal emphysema. There must, however, be an associated respiratory tract obstruction and sufficient respiratory effect on the part of the patient to aspirate air into the superior mediastinum. Without the respiratory tract obstruction this complication should not occur. The mediastinal air probably ruptures peribronchially into the pleural cavity. Once air has reached the pleural cavity any increased respiratory effort on the part of the patient will only result in a greater degree of pneumothorax. In the treatment of this condition, not only must the pneumothorax be corrected, but since an obstructed airway is the sine qua non of development its future patency must also be assured. We do not agree with the statement that the ingress of air into the superior mediastinum may be prevented by suturing the anterior and posterior layers of the carotid sheath over the ligated jugular vein stump.[2] As mentioned, the fascial layers are insufficiently well defined to prevent the ingress of air by their closure, and, second, the best insurance against further pneumomediastinum is a patent airway.

Tension pneumomediastinum has been mentioned as a possible cause of circulatory collapse after re-expansion of the lung. This has not been reported as an accompanying complication and would seem most unlikely in view of the wide communication between the superior mediastinum and surgical wound in the neck.

SUMMARY

Attention is called to the incidence of pneumothorax occurring during operative procedures about the neck and 4 cases reported without mortality. The development of this condition is dependent upon a partially obstructed airway in association with exposure of the superior mediastinum. The diagnosis should be entertained in any patient developing unexplained respiratory or cardiovascular difficulties in association with surgical procedures about the neck or in whom there may be a suggestion of ingress of air into the superior mediastinum. The principles of diagnosis and treatment are the same as those in any patient with a tension pneumothorax. Confirmation of the diagnosis should be made by immediate needle aspiration of both pleural cavities. Treatment consists of insertion of bilateral anterior thoracostomy tubes with underwater drainage.

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MIXED TUMORS OF THE PAROTID GLAND

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DESPITE the numerous studies that have been made on salivary tissue tumors, exact knowledge is still lacking. Of all the tumors incident to the parotid gland, the so-called mixed tumors are the most common and the most baffling. The modes of origin, biologic characteristics, and methods of treatment are subject to many divergent conceptions. This particular study of mixed tumors of the parotid gland is devoted to the correlation of clinical features and the results of specific treatment.

The designation "mixed tumor" will be retained in this study only because of its general acceptance and understanding by surgeons. However, this term is extremely vague and may actually be a misnomer. Willis[9] lists 15 different terms that have been used for the same entity. Adherence by pathologists to various theories of origin or composition has given rise to this welter of names. It is difficult and tedious to review the numerous theories that have been propounded in the last 90 years. Most of these have disintegrated with later and more accurate knowledge. Even today, there is no general acceptance of any one pathogenesis. Ahlbom[1] in his monograph on salivary tumors has the most detailed chronology of the history of salivary gland tumors. From his work is taken this brief exposition of current views. The idea that the mixed tumors originated in the parenchyma of the salivary glands appeared first in France and is still widely held. Most of those who do not adhere to this concept at least agree that the tumors are epithelial in origin. The mesenchymal and endothelial origin ideas of most German writers are now mainly of historical interest. The various applications of the Cohnheim theory of embryonic anlagen or superfluous cell, which rests with a great growth potential, still have many adherents.

The known vagaries in the growth potential of mixed tumors of the parotid gland require an alteration in our treatment criteria. The usual, although not universally accepted, idea that a 5-year follow-up of tumor patients is adequate for statistical study is inapplicable for analysis of mixed salivary tissue tumors. MacFarland[4] has shown that the average time interval between primary treatment and first recurrence is 7.2 years, and up to 30 years is not uncommon. Kirklin and associates[3] found that in 3 years following operation for primary mixed tumors 6.4 per cent had recurred. There was a linear relationship between the recurrence rate and the years postoperation, and at 5 years the recurrence rate was 22.5 per cent. Second recurrences also follow this same pattern, but

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at a higher rate. Such statistics therefore raise the question of the validity of conclusions based on studies with a follow-up of less than 5 years. Raising the minimum follow-up period above 5 years, except in unusual circumstances, is impractical due to the high case loss. All patients operated upon within the last 5 years have been excluded from this study. This exclusion has of necessity cut down on the statistical value of the series, but at least there will be less delusion about the results.

An attempt was made to have the patients return for personal examination, but the vast majority answered by completing a questionnaire. No case was included in which a reasonable question still existed about the tumor status during the observation interval. A definite pathologic examination and diagnosis of the tissue was necessary for inclusion. Because the cases spanned a 60-year period and pathologic diagnoses were made by several pathologists, some difficulty was encountered in the specificity of such diagnoses. This problem was partially solved by utilizing only those cases in which the diagnosis included the term "mixed tumor" or, in the questionable cases, where the microscopic description was obviously compatible with such a diagnosis.

The problem that this study hoped to clarify was the relative merits of the present treatment techniques (especially surgical) in preventing recurrences in this benign tumor group. Many surgeons performed the operations in this series of cases, and, as expected, the operative notes which were carefully perused followed no standard format or nomenclature. It was also evident that the extent of surgery varied within wide limits. We recognize three main types of surgical techniques. (1) Enucleation is the simple extirpation of the tumor with its capsule. (2) Excision is the en bloc removal of the tumor with a zone of normal tissue surrounding it. In this group the superficial lobectomies are placed, because it has been shown by several authors that the parotid gland is essentially unilobar and any procedure embodying a superficial lobectomy is a misnomer for a simple excision. (3) Total parotidectomy,* the most complete of the conservative procedures, is self-explanatory.

Sixty-five cases are included in this study, but the totals in the tables and figures in the text vary. The apparent discrepancy is due to chart lapses or vagueness on specific points.

The sex incidence was found to be without significance. There were 30 men in the series (46 per cent) and 35 women (54 per cent). The gland incidence appeared to be almost equilateral; the right side was involved in 31 cases and the left side in 34 cases.

The average age at onset for all cases in this series was 36.1 years. Between the ages of 20 and 50, 74.5 per cent of the tumors began (Table I). Again the age-sex difference was negligible, being only 0.2 years. It is of interest to note that in the cases that subsequently recurred, the onset age was less—31.7 years. Where there was no recurrence, the average onset age was 38.7 years. It was also noted that the recurring cases revealed significantly shorter onset-to-treatment durations. The average tumor duration in all cases was 5.3 years.

*Total parotidectomy as used in this paper means removal of the superficial and deep portions of the gland with preservation of the facial nerve. A few isolated acini may remain here and there. Obviously this is not an en bloc dissection as is necessary in malignant disease with consequent loss of the nerve.

In the cases that subsequently recurred, this time was 3.9 years, and in the nonrecurring cases, it was 6.2 years. (Table II.)

TABLE I. AGE AND SEX INCIDENCE

ONSET AGE	MALE		FEMALE		TOTAL (PER CENT)
	NUMBER	PER CENT	NUMBER	PER CENT	
0-10	3	4.5	0	0	4.5
10-20	1	1.5	3	4.5	6.0
20-30	7	11.0	11	17.0	28.0
30-40	9	14.0	8	12.5	26.5
40-50	4	6.0	9	14.0	20.0
50-60	4	6.0	2	3.0	9.0
60-70	0	0	1	1.5	1.5
70-80	1	1.5	1	1.5	3.0
Unknown	1	1.5	0	0	1.5
Total	30	46.0	35	54.0	100.0

TABLE II. ONSET AGE AND DURATION OF SYMPTOMS

	ONSET AGE		DURATION OF SYMPTOMS	
	NUMBER	AVERAGE AGE	NUMBER	AVERAGE (YEARS)
Primary cured cases	40	38.7	39	6.2
Recurrent cases	24	31.7	24	3.9
All cases	64	36.1	63	5.3

The average size of the mixed tumors in this series was 3.7 cm. In the cases that did not recur following surgical removal, the average size was 3.8 cm. with 36 per cent of the lesions being larger than 4.0 cm. In the cases that subsequently recurred, the average size was 3.6 cm., and 38.5 per cent of the lesions were larger than 4.0 cm. (Table III.)

TABLE III. APPROXIMATE SIZE OF LESIONS

	NONRECURRING	RECURRING
Cases	39	13
Average size (cm.)	3.8	3.6
Number of cases larger than 4 cm.	14 (36%)	5 (38.5%)

In 60 cases, surgical procedures were performed as primary therapy and were considered adequate by the operators. In 8 cases the operative notes were vague as to which technique was actually used. In each of these the question was between enucleation and excision. It would appear that the circumstances were unusual and the operators therefore confused as to the extent of the resection. The extremely high recurrence rate (62.5 per cent) in this group would tend to indicate that incomplete removal was common.

RESULTS

Where complete enucleation was performed primarily, recurrences were recorded in only 28 per cent. By adding secondary enucleations to the primary ones, the recurrence rate for all enucleation procedures then becomes 32 per cent. Excision of primary tumors resulted in a recurrence rate of 25 per cent and by adding the secondary operations the rate becomes 28 per cent. Total parotidectomy has come into wider use in this institution too recently for inclusion of many cases in this study. Only 5 cases were found for both primary

and recurrent tumors, but there were no recurrences following this procedure, even though the 3 secondary cases have been followed for 17, 17, and 20 years, respectively. Where irradiation alone was used therapeutically, the results were nil—100 per cent recurrence. Table IV shows the various combinations used for treatment and the results.

TABLE IV. RESULTS OF TREATMENT OF SUPPOSEDLY ADEQUATE SURGICAL THERAPY

TECHNIQUES	PRIMARY CASES			SECONDARY CASES			COMBINED RECURRENCE RATE
	TOTAL	RECURRENCES	PER CENT	TOTAL	RECURRENCES	PER CENT	
? Enucleation }	0	0	0	0	0	0	0
? or excision }	8	5	62.5	0	0	0	62.5
Enucleation	32	9	28.0	5	3	60.0	32.0
Excision	16	4	25.0	9	3	33.3	28.0
Enucleation with x-ray	2	0	0	0	0	0	0
Total parotidectomy	2	0	0	3	0	0	0
Total	60	18	30.0	17	6	35.3	31.6

Before assessing values to the different surgical techniques, the facial nerve status must be clarified. With wide dissection of the parotid area, as in total parotidectomies, it is to be expected that the facial nerve will be involved in more accidents than in the less manipulative procedures. Table V confirms this idea. Enucleation was followed by a permanent paralysis in 5 per cent, excision in 16 per cent, and total parotidectomy in 20 per cent of the cases. However, accidental division of the main trunk of the nerve was seen in 100 per cent of the cases of paralysis following enucleation and in 25 per cent of the excisions; whereas, total parotidectomies had no such catastrophes.

TABLE V. ACCIDENTAL PERMANENT FACIAL PARALYSIS FOLLOWING SURGERY

OPERATION	TOTAL CASES	PARALYSIS		PER CENT OF PARALYSES	PER CENT OF COMPLETE PARALYSES
		PARTIAL	COMPLETE		
Enucleation	39*	0	2	5.0	100.0
Excision	25	3	1	16.0	25.0
Total parotidectomy	5	1	0	20.0	0

*Includes 2 cases of enucleation and x-ray.

DISCUSSION

The high recurrence rate of mixed tumors of the parotid gland following supposedly adequate therapy is a challenge to all surgeons. In a recent paper,[10] we have reported investigations of the anatomy of this region in situ and have correlated the embryologic growth with that anatomy. The conclusions that the gland was irregular and unilobar confirmed the idea that present-day treatment, short of total parotidectomy, is actually inadequate treatment in a large percentage of cases. Many authorities already accept this thesis, and more extensive dissections are being performed. Too often, however, it is written that the superficial lobectomy is adequate for a tumor in the superficial part of the gland. The same defense has been made in previous decades for enucleation and excision procedures. However, perusal of recent articles on this subject reveal that the lowest recurrence rate for enucleation or excisions is about 10 per cent. The

logical conclusion to be drawn from the facts is that if all the potential tumor tissue were removed there could be no recurrence. It was decided to test this supposition by studying the results of various forms of treatment. Fortunately, the present study is one of several recent ones. By patterning the investigation somewhat on the others, it was hoped that the corresponding data might be merged and thereby increase the statistical validity of them.

Most series are in agreement that there is equal distribution between the sexes and between the right and left parotid glands. The onset age is quite uniform in most reported studies. It is predominantly a lesion of young adult life. Marshall and Miles's[6] series revealed the average onset age to be 34.3 years and the average tumor duration to be 10.6 years. McFarland, in 1943,[4] suggested that the recurrence rate of mixed tumors would be less if they were allowed to grow to a large size. He felt that they developed from multicentric foci and as the nodules grew larger they would coalesce into a single mass facilitating complete excision. In itself, the idea is not illogical and indeed many persons subscribed to it. We have two main reasons for not accepting this theory. First, our results show that the tumor size is almost exactly the same in the group that recurred as it is in the group that did not (Table III). Second, the larger tumors tend to make for a more difficult dissection with increased danger of facial nerve injury and spillage of tumor tissue into the field. Also Marshall and Forse[5] found that whereas benign mixed tumors had an onset-to-treatment time of 5.5 years, those cases where the mixed tumor contained areas of carcinoma the duration of symptoms was 9.6 years. Although the basis used for determining the size of the primary tumor is a mathematical estimate of the size as stated on the chart, the similarity in results is striking. It appears more likely that the greater recurrence rate in the shorter duration cases was due more to the rapid growth potential than to a size function.

The high recurrence rate (30.0 per cent) following supposedly adequate surgery is confirmed in this study (Table IV). Buxton and associates'[2] series revealed a corresponding rate of 10.1 per cent. This divergence of results is difficult to equate. However, a similar trend in the recurrence rate is noted when this total figure is broken down into the different operations performed. In the respective series it is found that enucleation procedures are followed by the highest recurrence rates. Excisional operations have a high rate of recurrence also, but slightly less than enucleations. Total parotidectomies in both series, although in much smaller numbers, were followed by no recurrences. It should be noted that in both series there is relatively little difference in the recurrence rates following enucleations and excisions. Only total parotidectomies show a marked improvement in recurrence rates (Table VI).

The incidence of permanent accidental facial paralyses showed a direct relationship to the extent of dissection (Tables V and VI). Temporary paralysis is not under consideration here, since it is seen to some degree in every case of parotid manipulation. It should be considered a physiologic sequela to parotid surgery rather than a complication. Whereas our series showed an incidence of both complete and incomplete paralyses of 5 per cent for the enucleations and 16 per cent for the excisions—a total of 9.5 per cent. Buxton's

TABLE VI. CORRELATED SERIES

SERIES	NO. CASES	RECURRENCES	PER CENT
Recurrences With Enucleation			
Buxton et al.	144	15	10.6
Winsten and Ward	37	12	32.0
Total	181	27	15.0
Recurrences With Excision			
Buxton et al.	40	4	10.0
Winsten and Ward	25	7	28.0
Total	65	11	16.9
Recurrences With Total Parotidectomy			
Buxton et al.	11	0	0
McCune	27	2	8.0
Winsten and Ward	5	0	0
Total	43	2	4.6

SERIES	NO. CASES	PARALYSES		PER CENT OF ALL PARALYSES	PER CENT OF PARALYSES THAT WERE COMPLETE
		PARTIAL	COMPLETE		
Permanent Paralyses With Enucleation and Excisions					
Buxton et al.	183	7	1	4.5	12.5
Marshall and Miles	92	7	3	10.9	30.0
Winsten and Ward	72	6	3	12.5	33.3
Total	347	20	7	7.8	35.0
Permanent Paralyses With Total Parotidectomy					
Buxton et al.	11	3	0	27.0	0
McCune	27	4	0	15.0	0
Winsten and Ward	5	1	0	20.0	0
Total	43	8	0	18.6	0

series showed a corresponding incidence of 4.5 per cent. Marshall and Miles, whose operation would be considered an excision by our standards, reported facial paralysis in 10.9 per cent of their series. In total parotidectomies the series revealed a 20 and 27 per cent paralysis incidence, respectively. It is of interest to note that McCune's[7] series of total parotidectomies showed only 15 per cent of facial nerve injuries. However, his recurrence rate was 8 per cent compared to the 0 per cent of both other series. The increased recurrence rate and decreased paralysis incidence might mean that the technique used in cases reported by McCune involved a less extensive dissection of the parotid area. This reasoning might well be extended to explain the difference between the Buxton figures and our own. It must be noted here that in all the series dealing with total parotidectomies, with the concomitant extensive dissection, no total paralyses are recorded.

The above statistics when carried to their logical conclusions raise an important philosophic question. Is the higher facial paralysis rate balanced by the lower recurrence rate from total parotidectomies? Kirklin and co-workers[3] hold that simple excisions are justified because the life expectancy statistics are not altered by the presence of mixed tumors and less extensive procedures have a much lower nerve accident rate.

The arguments against this approach are also compelling. Secondary operations have been shown to be more hazardous to the facial nerve than primary ones. The Marshall and Forse[5] series recorded a 6.1 per cent facial nerve

injury (extent not specified) in primary cases, but secondary operations were followed by a 27.5 per cent palsy incidence. Also, tumors that recur are more prone to further recurrence than primary tumors. The possibility of malignant growth developing in the recurrence has been stressed by Slaughter and associates.[8] Facial nerve accidents in total parotidectomies, although more common, are mostly of the single branch type. Rarely does injury occur to the main trunk, probably because of adequate exposure of the nerve. In the less extensive procedures where exposure and isolation of the nerve plexus is not so fundamentally necessary, fewer facial nerve injuries occur, but, of those that do, more are complete divisions. Once the nerve plexus has been exposed, removal of the deep portion of the parotid gland can be effected with relative ease and safety.

CONCLUSIONS

From the available statistics in the medical literature and our present series, it is evident that the conservative methods of therapy for mixed tumors of the parotid gland are inadequate for permanent cure. Even a cure rate of 90 per cent should not be acceptable, and most surgeons do not obtain even this result. Total parotidectomy, where practically all tumor-bearing tissue is removed, yields the highest cure rate.

Early operation for mixed tumors is necessary for the following reasons: (1) Complete removal is more easily accomplished when the lesions are smaller, and (2) early removal reduces the chance of malignant change. It is hoped that in the future more such operations will be performed and reported in order to accurately evaluate these tenets. Statistics also point to a lower percentage of complete facial paralyses following total parotidectomy than following the less radical operations of enucleation and excision.

SUMMARY

A study of the literature dealing with end results in the treatment of mixed tumors of the parotid gland and 65 cases taken from the files of The Johns Hopkins Hospital have been correlated and presented.

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THE CASE FOR THE INFRADIAPHRAGMATIC REPAIR OF ESOPHAGEAL HIATAL HERNIA

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THE routes of approach for the surgical repair of hiatal hernias have not been as varied as women's hats, but certainly dictated by as few authorities.

In the United States the outstanding clinical authority referable to hiatal as well as all types of diaphragmatic hernias has been Stuart Harrington.[4] The overwhelming popularity of the abdominal approach up until a decade ago was due largely to this individual's clinical stature and teaching. However, with the maturing of thoracic surgery, there was a steady swing to the transthoracic approach. The popularity of this surgical route reached complete dominance with the aid of Sweet[7, 8] in the United States and Allison[1] in England.

In the last few years there has been a swing back to the almost completely abandoned abdominal approach.[2] We would like to add our clinical experience in support of this trend.

ANATOMY AND PHYSIOLOGIC CONSIDERATIONS

Although the terminal esophagus does not possess a true anatomic sphincter comparable to the anal or pyloric sphincter, it does contain a functional sphincter mechanism, the chief purpose of which is to prevent regurgitation of damaging juices from the stomach into the lower esophagus. This is referred to as the cardiac sphincter mechanism and is composed of the following parts:[6] (1) the constrictor cardia (this is not anatomically demonstrable but is physiologically present), (2) the phrenoesophageal membrane, which joins the esophagus to the diaphragm (it arises from the undersurface of the diaphragm and inserts into the esophagus at the level of the inferior esophageal sphincter superiorly and into the cardia of the stomach inferiorly), (3) a valve action dependent upon the obliquity of the junction between the esophagus and the stomach (in deep inspiration this angle changes and the lower esophagus is, therefore, pinched off), and (4) the pinch-cock action of the diaphragm resulting from the contraction of the diaphragmatic fibers of the crus surrounding the hiatus.

The right diaphragmatic crus and the phrenoesophageal ligament combine to maintain the stomach below the diaphragm and to maintain the acute angle of entry of the esophagus into the stomach.

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The hiatus, which is directly exposed to the pressure variation between thoracic and abdominal cavity, is the weakest point in the diaphragm and the most frequent site of herniation. The esophageal hiatus is anterior and slightly to the left of the aortic opening, and it is formed by the decussating fibers of the right and left crura. It is more truly a split in the muscle fibers of the right crus reinforced by fibers from the left. The right crus forms a sling around the esophagus, and this sling is fixed inferiorly by the attachment of the crus to the vertebral column. Allison has compared this sling to that found in the pelvis where the puborectalis lassoes the rectum.

The esophagus anteriorly at the diaphragm is supported by transverse muscle fibers continuous on each side with the perpendicular fibers of the crus, decussating with one another to form a reinforced raphe. Posteriorly, there is less support because it is here that the crura split to form the hiatus. When the esophageal hiatus is enlarged, the pressure applied in front of the esophagus and at the sides is acting perpendicular to the muscle fibers "across the grain" and may only cause some atrophy of the muscle fibers, but little more. The pressure posteriorly, however, is acting in the same direction of the muscle fibers "along the grain" and splits the fibers to increase the size of the hiatus. This enlargement of the hiatus posteriorly is the main defect present in sliding hiatal hernia.

In normal breathing the diaphragm serves as a barrier at the lower border of the chest to preserve the changes in pressure caused by the motion of the chest wall. During inspiration, when the ribs are elevated, the central tendon of the diaphragm is stabilized by the muscular contraction of the diaphragm and the dome becomes flattened and depressed. During expiration the musculature of the diaphragm is relaxed and it resumes its domelike contour, which is further exaggerated by the downward and inward motion of the ribs and the contraction of the abdominal wall. When the diaphragm contracts, the action of the right crus on the cardia is twofold. It compresses the wall of the esophagus from side to side and it pulls down and increases the angulation of the esophagus. In addition, the contraction of the diaphragm produces tension of the phrenoesophageal membrane and, by its attachment to the esophagus, stabilizes the lower esophagus. It should be noted that it is during inspiration, when the suction in the chest combined with the increased abdominal pressure would tend to draw the gastric content into the esophagus, that the crura of the diaphragm contract and obstruct this tendency toward regurgitation.[3]

The position of the cardia depends partly on the muscular action of the diaphragm and partly on the ligamentous attachments. The deep fascia on the undersurface of the diaphragm (the phrenoesophageal membrane) passes through the hiatus to become continuous with the fascia propria of the esophagus. This means that the pull of the diaphragm over the esophagus in inspiration is more evenly distributed over the organ, instead of being concentrated only at the cardia.

TYPES OF HIATAL HERNIAS

Hernias through the hiatus are of 2 main varieties, (1) the sliding type and (2) the parahiatal (paraesophageal). Infrequent variants are (1) the congenitally short esophagus and (2) double hernia (hiatal and parahiatal).

The majority of herniations through the diaphragm are of the sliding type and constitute from 80 to 90 per cent of the cases in which patients come to surgical treatment. In our series of 20 consecutive surgical cases during the past 1½ years, all hernias were of this type. This type is comparable to direct inguinal hernias since both are due to weakness of the region and do not develop in a preformed sac. In this type of hernia, the acute angle between the esophagus and stomach disappears. The cardia slides up in the mediastinum, taking the phrenoesophageal membrane and peritoneum with it. The esophagus recoils so that it appears shorter, but may be pulled back by slight traction at operation, showing that the esophagus is relaxed but not actually short. Patients with this type of hernia are prone to regurgitate digestive juices from the stomach into the esophagus and to develop esophagitis.

Parahiatal (paraesophageal) hernia is a less frequent type and constitutes approximately 10 per cent of surgically treated cases. In this type the esophagus has maintained its attachment to the diaphragm and the cardial end of the stomach has herniated through the abdominal opening alongside of the esophagus. The esophagus retains its normal length and the cardia remains below the diaphragm. This type of hernia is comparable to the indirect inguinal hernia.

It is possible that in some individuals, during development when the stomach prolapses into the peritoneum from behind, a protrusion of peritoneum may remain in the hiatus, much like in the inguinal region. Since the prolapse of the stomach is from behind, if a protrusion of the peritoneum remains in the mediastinum it must occur in front. This preformed sac may remain empty and small or may, under certain circumstances such as aging, obesity, increased abdominal pressure, sudden weight loss, and so forth, enlarge and the anterior surface of the stomach roll in, dragging along, in severe cases, omentum and colon. In spite of this displacement, the esophagus still enters the stomach at an acute angle; furthermore, when the hernia enlarges it compresses the esophagus from the anteroposterior direction. Regurgitation of gastric content in the esophagus and esophagitis do not occur.

CLINICAL CONSIDERATIONS

The clinical material represents our experience in the surgical treatment of esophageal hiatal hernias from October, 1955, to March, 1957, at the Veterans Administration Hospital. During this period of time, 41 patients were discharged with the diagnosis of esophageal hiatal hernia, of which 21 were treated by medical management and 20 underwent surgical repair. All 20 patients had the sliding variety of hernia (Table I).

TABLE I

PATIENT	AGE	SYMPTOMS	DURATION OF SYMPTOMS	REPAIR	POSTOP. HOSPITAL DAYS	MORTALITY	COMPLICATIONS
M. R.	35	Anemia—melena; hematocrit 20 per cent	2 weeks	Thoracic	5	0	0
N. W.	66	Esophagitis, gaseous duodenal ulcer symptoms, gall bladder symptoms	20 years	Abdominal	12	0	0
O. E.	37	Burning epigastric pain, melena, 2 occasions	12 years	Abdominal	6	0	0
N. H.	38	Acute upper abdominal pain, epigastric and lower chest pain	2 weeks	Abdominal	6	0	0
H. A.	42	Regurgitation; esophagitis	6 months	Abdominal	5	0	0
A. W. T.	62	Esophagitis, upper gastrointestinal bleeding, regurgitation	5 years	Thoracic	25	0	Intercostal neuroma
A. W. T.	62	Esophagitis, epigastric and lower sternal pain	6 months	Abdominal	8	0	0
T. G.	39	Esophagitis, dysphagia, liver disease, portal circulation, gastrointestinal bleeding	10 years	Thoracoabdominal	14	0	0
F. M.	41	Epigastric postprandial pain	4 years	Abdominal	6	0	0
McR.	55	Upper abdominal pain, vomiting, weight loss 70 pounds	2 weeks	Thoracic	15	0	0
L. E.	66	Epigastric pain, vomiting	1 week	Abdominal	8	0	0
J. M.	62	Epigastric and upper quadrant pain, hiccough	13 years	Abdominal	12	0	0
E. P.	58	Dysphagia, hematemesis, minor	4 years	Abdominal	12	0	0
G. L.	43	Heart burn, regurgitant esophagitis	10 years	Abdominal	8	0	0
B. E.	60	Epigastric and retrosternal pain	2 years	Abdominal	5	0	Incisional hernia
M. F.	64	Vomiting, bleeding, epigastric pain	10 years	Abdominal	13	0	0
S. N.	60	Melena, upper quadrant pain, gastrointestinal bleeding, esophagitis, regurgitation	6 months	Abdominal	7	0	0
D. R.	59	Melena, epigastric pain, regurgitant esophagitis	3 years	Abdominal	10	0	0
M. J.	74	Substernal pressure, postprandial indigestion, syncope	10 years	Thoracic	7	0	0
M. F.	61	Hematemesis, melena, anemia, hematocrit 23 per cent	4 months	Abdominal	8	0	0

Four procedures were performed through the thoracic approach, 15 through the abdominal, and one through a thoracoabdominal incision.

The mean average age of these patients was 50.3 years, ranging from 34 to 74; the most common occurrence was in the fifth and sixth decades of life. The duration of symptoms varied from 2 weeks to 14 years.

The average period of postoperative hospitalization was 8 days when the abdominal approach was used, and 13 days following the thoracic route. There was no mortality in this series and only 2 postoperative complications, one incisional ventral hernia in the abdominal series which was later repaired and one intercostal neuroma in the thoracic series which required excision twice. Esophagitis confirmed by biopsy and/or esophagoscopy was found in 10 cases (50 per cent). Ten patients (50 per cent) had either melena, hematemesis, or history of gastrointestinal bleeding.

Since this series is very recent, we felt that a follow-up in regard to recurrence is of little or no value.

The symptoms of esophageal hiatal hernia so closely resemble those of cardiac respiratory or other gastrointestinal diseases that many patients with hiatal hernia are often treated for simulated conditions for a long time before the correct diagnosis is made.

Harrington, in a report of 343 cases,[5] has found that an average of 3 erroneous diagnoses had been made before the correct one was established. The most common erroneous diagnoses were cholecystitis, cholelithiasis, gastric or duodenal ulcer, hyperacidity, and anemia. We also have been impressed in our series by the high incidence (over 50 per cent) of associated pathologic conditions in the presence of a hiatal hernia (Table II).

TABLE II

ASSOCIATED CONDITIONS	
Gastric ulcer in hernia	1
Duodenal ulcer, old	1
Early cirrhosis	1
Cholelithiasis	2
Meckel's diverticulum	1
Duodenal ulcer and pyloric stenosis	2
Carcinoma of pancreas	1
EXCLUDED CONDITIONS	
Duodenal ulcer	
Nonfunctioning gall bladder	
Esophageal varices	

The symptoms produced by the esophageal hiatal hernia and the ones due to additional intestinal tract pathology often cannot be distinguished or diagnosed until the abdominal cavity has been adequately explored. In the 15 cases where the abdominal cavity was adequately explored, 12 concomitant or associated conditions were found and 3 conditions diagnosed or suspected by x-ray findings were excluded. In one case the presence of an inoperable carcinoma of the pancreas which was completely unsuspected has been established by exploration and biopsy. It is quite possible that the condition would not have been recognized if we had repaired the hiatus via the thoracic route, and this patient would have required an additional surgical procedure.

As a corollary, 7 of the 15 patients who had transabdominal repair of the hiatus had additional intra-abdominal surgical procedures which would have been very difficult and, in some cases, impossible to perform through the thoracic incision (Table III).

TABLE III. ADDITIONAL SURGICAL PROCEDURE

PROCEDURE		INDICATION
Meckel's diverticulum excision	1	
Cholecystectomy	2	Cholelithiasis
Liver biopsy Portal pressure	1	Early cirrhosis ? of esophageal varices
Vagotomy and gastroenterostomy	1	Pyloric stenosis
Vagotomy and pyloroplasty	1	Pyloric stenosis
Cholecystojejunostomy	1	Carcinoma of pancreas

TECHNICAL CONSIDERATIONS

A study of the functional anatomy of the esophageal hiatus reveals that a physiologic repair requires (1) division of the phrenoesophageal membrane and its subsequent foreshortening and reattachment; (2) reduction of the herniated viscera; (3) closure of the hiatal defect with preservation of the cardiac sphincter mechanism; (4) re-establishment of the acute angle of entry of the esophagus into the stomach; (5) preservation of the pinch-cock action of the diaphragm resulting from the contraction of the muscle fibers of the crura during inspiration; and (6) closure of the hiatal defect by nonstrangulating sutures to approximate the muscle fibers of the crura behind the esophagus.

In our experience the abdominal approach satisfies best these conditions for a physiologic repair. Division of the phrenoesophageal membrane is done under direct vision by sharp dissection and subsequent foreshortening and reattachment is easier. Reduction of the hernia is done with ease by gentle traction at the esophagogastric junction.

Both crura of the diaphragm are easily exposed by this approach and approximated behind the esophagus by a few sutures and the normal anatomic and functional position of the gastroesophageal junction with preservation of the sphincter mechanism is re-established. A glance at the anatomy of the diaphragm shows that to close the hiatus at the sides ruins the very muscle upon which function depends, and to close it in front merely displaces the esophagus posteriorly and the acute angle of entry, as well as the pinch-cock action of the crura, is lost. We do not recommend the abdominal approach when a short esophagus is suspected. If such a condition is found (1 to 2 per cent), the abdominal incision should be extended into the chest.

CONCLUSION

We advocate the abdominal approach for the repair of esophageal hiatal hernia because it allows adequate exploration of the abdominal cavity—a consideration of importance since most patients having esophageal hiatal hernias are usually of an advanced age and frequently present symptoms of other gastrointestinal pathology, it permits more adequate performance of other intra-abdominal operations, it affords the best exposure of both diaphragmatic crura and phrenoesophageal membrane with easier and more adequate repairs, and it satisfies best the criteria for anatomic and functional repair of the hiatus.

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THE INTESTINAL FACTOR IN IRREVERSIBLE HEMORRHAGIC SHOCK

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ISCHEMIC injury to the liver of the dog occurring during prolonged hemorrhagic shock has been thought by many investigators to be the basic cause of irreversibility following prolonged hemorrhagic shock.[1, 2, 8, 10] However, the striking lesions of irreversible hemorrhagic shock at autopsy are mucosal congestion and necrosis of the small and large bowel.[21] Despite this fact, no direct attempts to prevent these bowel lesions from occurring during hemorrhagic shock have been made. Rather, attention has been focused on methods to increase blood flow to the liver during shock[1, 2, 8, 10] or on altering the general state of experimental animals prior to shock with adrenolytics[12, 18] vasodilators,[9, 11, 20] or hypothermia.[14, 17] To determine whether or not the bowel lesions were of primary importance in the genesis of irreversibility, a procedure was developed to maintain the superior mesenteric arterial flow of dogs in shock at normal levels. The results of these experiments were compared with results of other experiments in which similar amounts of arterial blood were introduced into the general systemic arterial or venous circulation or into the liver or brain of dogs in shock.

METHODS

All experiments were performed in an air-conditioned room and utilized unanesthetized mongrel dogs sedated with morphine sulfate, 2 mg. per kilogram, intramuscularly. All dogs were fasted for 18 hours prior to an experiment but were allowed water until sedated.

Group I. Superior Mesenteric Arterial Perfusion.—In the dog to be bled, No. 14 F. polyvinyl catheters were placed in the superior and inferior venae cavae via the external jugular and femoral veins. Another No. 14 F. catheter was placed in the femoral artery for bleed-out and retransfusion. An unanesthetized donor dog was readied with similar catheters in the aorta and inferior vena cava inserted via the left femoral artery and vein. A third catheter was placed in the right femoral artery of the donor dog to measure blood pressure. All cannulations were done using local piperocaine hydrochloride anesthesia. Both experimental and donor dogs were given heparin,

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2 mg. per kilogram, intravenously at the time the cannulations were made. The donor dog was given a second similar dose of heparin intravenously midway through the subsequent perfusion.

Two hours following sedation, the experimental dog was hemorrhaged to 35 mm. Hg mean arterial pressure, utilizing a new automatic leveling device and blood reservoir for the control of hypotension (Fig. 1).[4] When maximal bleed-out was reached (1.5 ± .48 hours in 60 previous hemorrhage experiments in this laboratory), the superior mesenteric artery of the shocked dog was isolated through a left flank retroperitoneal approach. This artery was

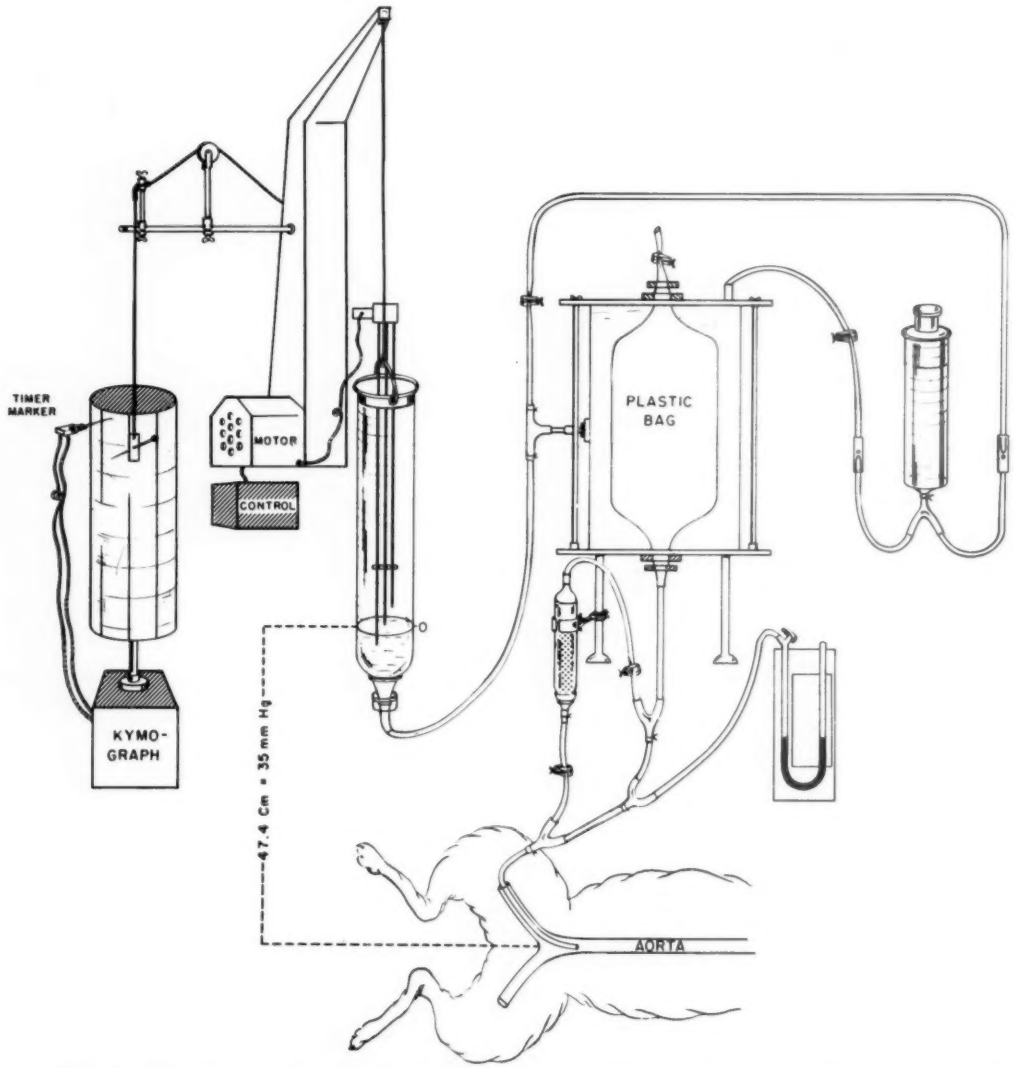

Fig. 1.—The dog bleeds into a limp plastic bag enclosed in a water-filled tank. The blood displaces an equal amount of water into the open cylinder. Sensing electrodes connected to an electronic control maintain the water surface in the cylinder at 47.4 cm. (equals 35 mm. Hg) above the aorta of the dog, regardless of the bleed-out or uptake volume, by means of a reversible motor which can raise or lower the cylinder. Changes in bleeding volume are recorded on a moving kymograph by means of a pulley system connected to the cylinder. By adjustment of clamps at the end of the experiment, water can be pumped back into the tank at any desired speed with the syringe. This forces blood back into the dog through the filter. A mercury manometer provides a constant reading of the dog's systemic arterial pressure. When maximal bleeding volume is reached, cross perfusion of the bowel via the superior mesenteric artery is started.

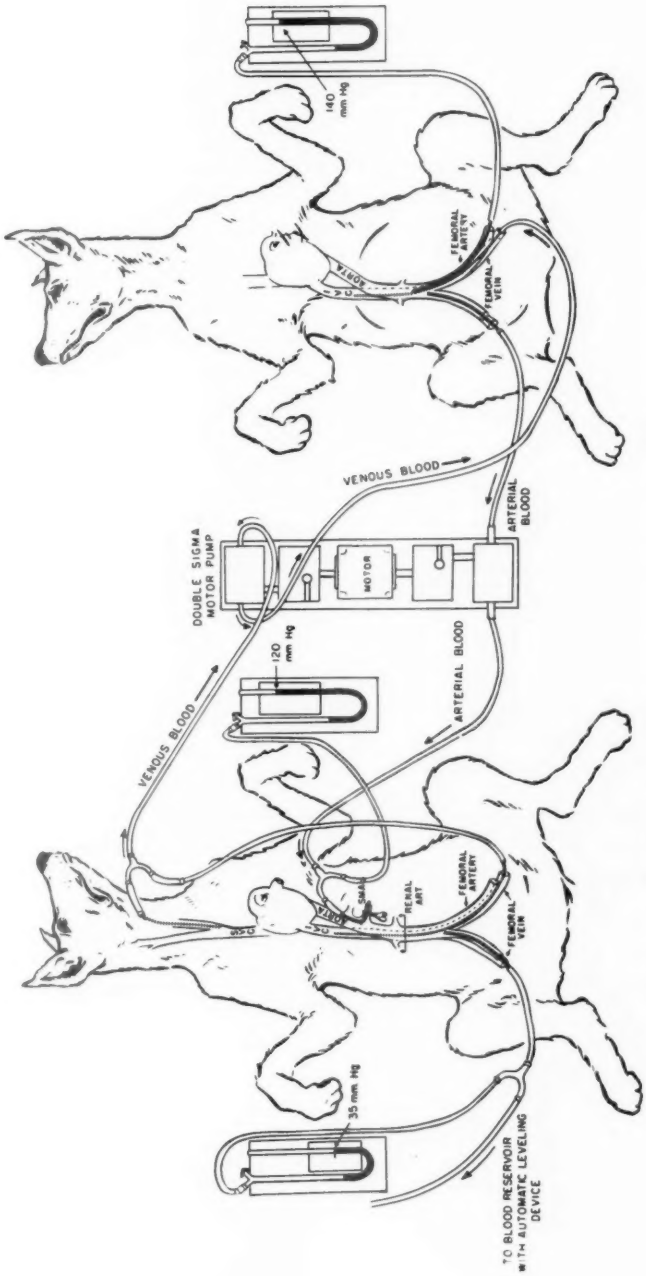

FIG. 2.—The experimental setup for perfusion of the superior mesenteric artery.

clamped off from the aorta and cannulated with a 3 mm. inside diameter siliconized glass cannula. The profound shock plus small amounts of local piperocaine hydrochloride gave ample anesthesia for this procedure which could be done in 15 minutes. Cross perfusion of the bowel via the superior mesenteric artery was then immediately started using a double Sigmamotor pump and arterial blood from the donor. Simultaneously, an equal amount of vena caval blood was returned from the shocked dog to the normotensive donor (Fig. 2). We have previously found that blood flow in the superior mesenteric vein of normotensive dogs ranged from 8 to 12 ml. per kilogram per minute. A flow in this range was used for perfusions in all dogs in this and subsequent groups. Throughout the period of perfusion, the shocked dog's mean systemic arterial pressure remained at 35 mm. Hg. Initially, the mean perfusion pressure in the superior mesenteric artery averaged 150 mm. Hg. However, although flow remained constant, the perfusion pressure in the superior mesenteric artery gradually increased to 200 mm. Hg by the end of the period in shock. The donor's systemic arterial pressure remained in the normal range, 150 mm. Hg (Fig. 3).

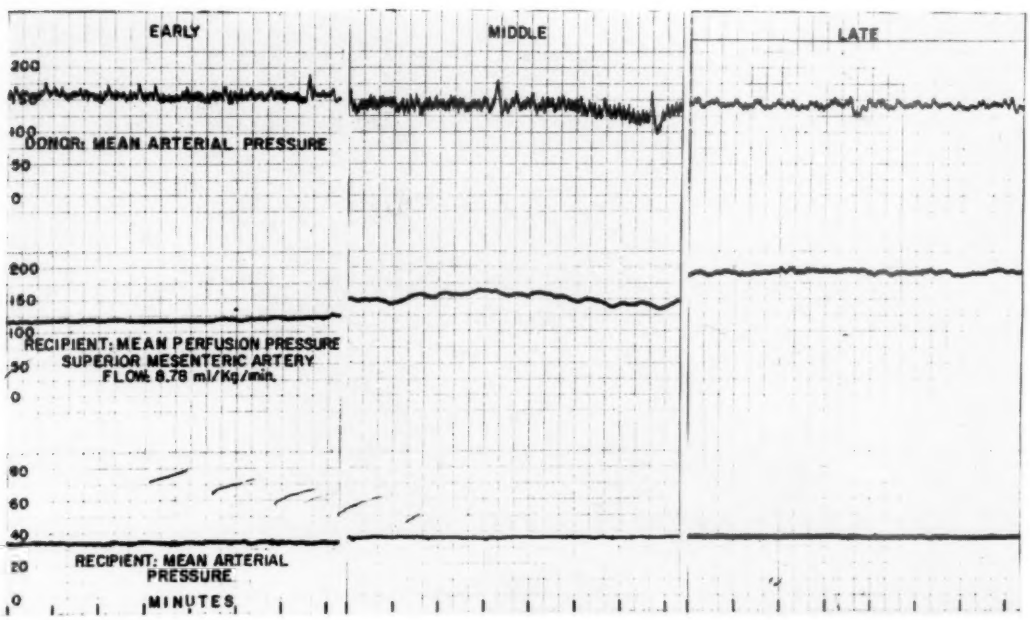

Fig. 3.—Mean arterial pressures in the donor and recipient dogs during perfusion of the superior mesenteric artery. Note the gradual increase in perfusion pressure in the superior mesenteric artery of the shocked dog.

Four and one-half hours after the start of bleeding, the perfusion was stopped, the cannula was removed from the superior mesenteric artery, and the artery was repaired with 6-0 silk and unclamped from the aorta. Then, the flank wound was rinsed profusely with sterile saline and closed while the dog was being retransfused with all shed blood in the reservoir about 5 hours from the start of hemorrhage.

Group II. Superior Mesenteric Arterial Perfusion in Eck Fistula Dogs.—End-to-side Eck fistulas were made in mongrel dogs through a thoracoabdominal incision using aseptic technique and pentobarbital anesthesia. One to 2 months later, these dogs were hemorrhaged as in Group I. They then received bowel perfusion via the superior mesenteric artery exactly as in Group I. In these dogs, perfusion pressure in the superior mesenteric artery averaged 130 mm. Hg and increased only slightly by the end of the shock period.

Group III. No Perfusion.—Normal dogs were hemorrhaged as in Group I. The superior mesenteric artery was then isolated but not cannulated. These dogs were held in shock for a period similar to that in Group I and retransfused.

Group IV. Inferior Vena Caval Perfusion.—Dogs were hemorrhaged as in Group I. The superior mesenteric artery was isolated but not cannulated. Instead, arterial blood from the donor was perfused into the inferior vena cava of the shocked dog via a femoral vein at 8 to 12 ml. per kilogram per minute, returning equal amounts of superior vena caval blood to the donor. Perfusion was stopped and the shocked dog was retransfused at about 5 hours following onset of hemorrhage.

Group V. Lower Abdominal Aortic Perfusion.—Dogs were hemorrhaged as in Group I. The superior mesenteric artery was isolated but not cannulated. Instead arterial blood from the donor was perfused into the lower abdominal aorta via a femoral artery at 8 to 12 ml. per kilogram per minute, returning equal amounts of vena caval blood to the donor. Perfusion was stopped and retransfusion effected about 5 hours following the onset of hemorrhage.

Group VI. Liver Perfusion Via the Celiac Axis.—Dogs were hemorrhaged as in Group I. The celiac axis was isolated from the aorta and cannulated through the same left flank retroperitoneal approach used for the superior mesenteric artery in Group I dogs. Again the anesthesia afforded by the shock was ample for this procedure which could be done in 15 minutes. Arterial blood from the donor was then directed into the liver of the shocked dog via the celiac axis at 8 to 12 ml. per kilogram per minute, returning equal amounts of vena caval blood to the donor. This flow gave a mean perfusion pressure of 130 mm. Hg in the celiac axis, and this pressure remained quite constant throughout the shock period. After 4½ hours of shock, perfusion was stopped, the cannula was removed from the celiac axis, and this artery was repaired with 6-0 silk. The celiac axis was then opened to the aorta. The wound was rinsed with saline and closed. Retransfusion was effected about 5 hours following the start of hemorrhage.

Group VII. Liver Perfusion Via the Portal System.—These dogs were splenectomized and the splenic vein transplanted to an accessible subcutaneous site; pentobarbital sodium anesthesia and aseptic technique[8] were used. No antibiotics were used postoperatively. Two to 4 weeks later all wounds were

healed. The dogs were then hemorrhaged as in Group I. The subcutaneous splenic vein was quickly isolated and cannulated. Again the shock itself gave ample anesthesia for this procedure. Donor arterial blood was then directed into the liver via the portal splenic artery at 8 to 12 ml. per kilogram per minute, returning equal amounts of vena caval blood to the donor. The portal pressure averaged 25 mm. Hg with this flow. Perfusion was terminated and the perfused dog was retransfused after 5 hours of hemorrhagic shock.

Group VIII. Brain Perfusion Via the Carotid Artery.—Dogs were hemorrhaged as in Group I. The right common carotid artery was then quickly isolated and cannulated. Perfusion of the brain was begun with donor arterial blood at a rate of 8 to 12 ml. per kilogram per minute, returning equal amounts of vena caval blood to the donor. Perfusion was terminated, and the shocked dog was retransfused about 5 hours following the onset of hemorrhage. Mean perfusion pressures in the carotid artery averaged 120 mm. Hg with this flow and remained quite constant throughout the shock period.

Blood cultures were taken from the plastic blood reservoir and from the shocked dog following retransfusion. Plasma volumes were measured in all dogs before hemorrhage and after retransfusion with a micromethod using T-1824, the injected dye being weighed and the plasma analyzed in microcuvettes.[13] Blood volumes were then calculated using the venous hematocrit. The perfusion pressure and systemic arterial pressure of the shocked dog were continuously recorded along with the systemic arterial pressure of the donor with Statham strain gauges of suitable sensitivity and a Sanborn Poly-Viso recorder. The blood reservoir, plastic tubing, and all cannulae were soaked in benzalkonium chloride for 24 hours and rinsed with sterile saline before use. Clean technique was used for all surgical procedures. All fatalities were autopsied. All survivors were sacrificed and also autopsied at varying periods after the experiment. Representative tissue specimens were taken for microscopic examination.

RESULTS

Prior to hemorrhage, there was no significant statistical difference (t test) between any of the groups of dogs in body weight, hematocrit, blood volume, or mean systemic blood pressure (Table I). Table II summarizes the results in the various groups of dogs. Only bowel perfused dogs in Groups I and II survived in significant numbers. In all other groups, most of the dogs died of irreversible hemorrhagic shock. Maximal bleeding volumes and the time to reach maximal bleeding volume were similar for all groups. Perfusion flows were also similar for all groups. Dogs receiving aortic perfusion or no perfusion were in shock somewhat less than 5 hours because they generally showed signs of excessive uptake and/or cardiorespiratory failure necessitating retransfusion before the prescribed period was completed.

In Groups I and II, it took a mean of 18 ± 3.9 minutes to repair the superior mesenteric artery at the end of the shock period. In Group VI, it took a mean of 19 ± 5.4 minutes to repair the celiac axis at the end of the shock period. Dogs were not considered survivors unless they were alive 72 hours

TABLE I. PREHEMORRHAGE VALUES*

GROUP	TYPE OF PERFUSION	NUMBER OF DOGS	BODY WEIGHT (KG.)	HEMATOCRIT	BLOOD VOLUME† (ML./KG.)	MEAN SYSTEMIC ARTERIAL BLOOD PRESSURE (MM. HG)
I	Superior mesenteric arterial	30	19.7 ±3.3	50.0 ±5.7	104.0 ±11.0	121.0 ±11.0
II	Superior mesenteric arterial, Eck fistula	10	16.1 ±3.6	48.3 ±3.3	99.6 ±6.9	107.0 ±9.8
III	None	15	20.0 ±5.1	50.4 ±5.3	109.0 ±15.0	133.0 ±15.0
IV	Inferior vena caval	10	20.4 ±5.0	50.8 ±6.0	107.0 ±13.0	118.0 ±7.4
V	Aortic	10	17.5 ±2.4	49.8 ±4.4	102.0 ±9.6	131.0 ±15.0
VI	Celiac axial	10	20.8 ±2.7	50.6 ±4.6	111.0 ±13.0	133.0 ±8.3
VII	Portal	10	17.5 ±2.7	43.4 ±6.1	88.3 ±13.0	125.0 ±12.0
VIII	Carotid	10	20.3 ±4.4	47.6 ±2.8	101.0 ±9.2	116.0 ±16.0

*Mean values with standard deviations.

†Blood volumes were not corrected for plasma trapping in the hematocrit tube nor for body hematocrit.

TABLE II. HEMORRHAGIC SHOCK AT 35 MM. HG MEAN ARTERIAL BLOOD PRESSURE*

GROUP	TYPE OF PERFUSION	NUMBER OF DOGS	NUMBER OF SURVIVORS	MAXIMAL BLEEDING VOLUME		PERFUSION		HOURS IN SHOCK	BLOOD VOLUME CHANGE PER CENT	HOURS DEATH SHOCK AFTER
				PER CENT BODY WEIGHT	PER CENT BLOOD VOLUME	START IN HOURS AFTER HEMORRHAGE	FLOW (ML./KG./MIN.)			
I	Superior mesenteric arterial	30	27	6.0 ±0.94	58.6 ±7.6	1.48 ±0.38	8.84 ±2.2	4.90 ±0.23	6.21 ±4.2	- -
II	Superior mesenteric arterial, Eck fistula dogs	10	9	5.09 ±0.39	51.5 ±3.9	1.01 ±0.28	9.52 ±2.3	5.0 ±0.20	7.33 ±6.3	- -
III	None	15	1	5.73 ±0.55	53.5 ±4.6	- -	- -	4.40 ±0.40	7.21 ±3.3	6.39 ±3.9
IV	Inferior vena caval	10	2	6.04 ±0.93	56.0 ±4.5	1.3 ±0.07	9.0 ±0.92	4.94 ±0.29	6.87 ±4.0	5.86 ±3.1
V	Aortic	10	2	5.78 ±0.64	57.3 ±5.6	1.32 ±0.19	10.9 ±2.4	4.64 ±0.20	7.60 4.6	7.03 ±6.1
VI	Celiac axial	10	4	6.37 ±0.90	57.8 ±7.3	1.37 ±0.23	9.86 ±0.72	4.97 ±0.14	4.98 ±5.1	12.5 ±6.8
VII	Portal	10	3	4.39 ±1.4	51.2 ±7.3	1.44 ±0.36	10.9 ±2.2	4.95 ±0.10	8.34 ±3.4	9.64 ±4.5
VIII	Carotid	10	2	5.13 ±0.79	53.9 ±8.0	1.25 ±0.19	8.72 ±0.83	4.92 ±0.06	7.81 ±6.9	26.2 ±4.0

*Mean values with standard deviations.

following the experiment. Dogs dying following the hemorrhagic shock manifested the usual signs of irreversibility. They had a recovery of blood pressure to normal levels upon retransfusion followed by a gradual decline with the onset of copious bloody diarrhea. At autopsy, these dogs exhibited severe mucosal congestion and necrosis of the small and large bowel along with marked liver congestion (Fig. 4). Dogs with irreversible shock in the various groups died in very similar average times with the exception of dogs receiving brain perfusion via the carotid artery in Group VIII. These dogs had a significantly prolonged survival (26.2 ± 4.0 hours, $p = .001$) although there was no increase in the ultimate number of survivors.

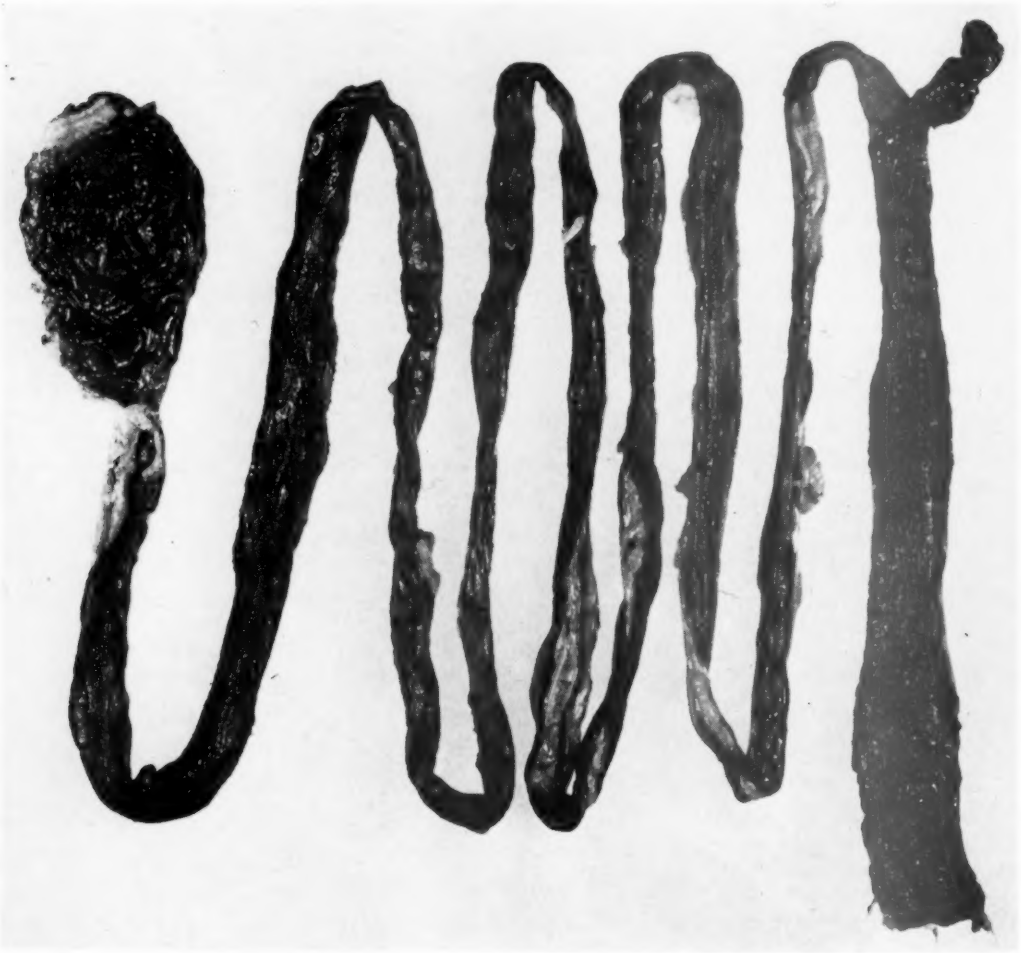

Fig. 4.—Stomach and intestine from dog with irreversible shock. Mucosal congestion and necrosis is present throughout the small and large bowel.

Surviving superior mesenteric arterial perfused dogs in Group I sacrificed at varying periods following the experiment had normal bowels (Fig. 5). The livers of these dogs, however, showed the same degree of congestion, both grossly and microscopically, as was seen in the livers of dogs in other groups

dying of irreversible hemorrhagic shock. Surviving dogs in Group II also were sacrificed and also had normal bowels. The livers of these Eck fistula dogs were not grossly or microscopically congested and showed only fatty infiltration, a common finding in the livers of chronic Eck fistula dogs. The gradual rise of perfusion pressure in the superior mesenteric artery in Group I dogs and the absence of any significant rise in superior mesenteric perfusion pressure in Group II dogs are possibly explained on the basis of these liver findings.

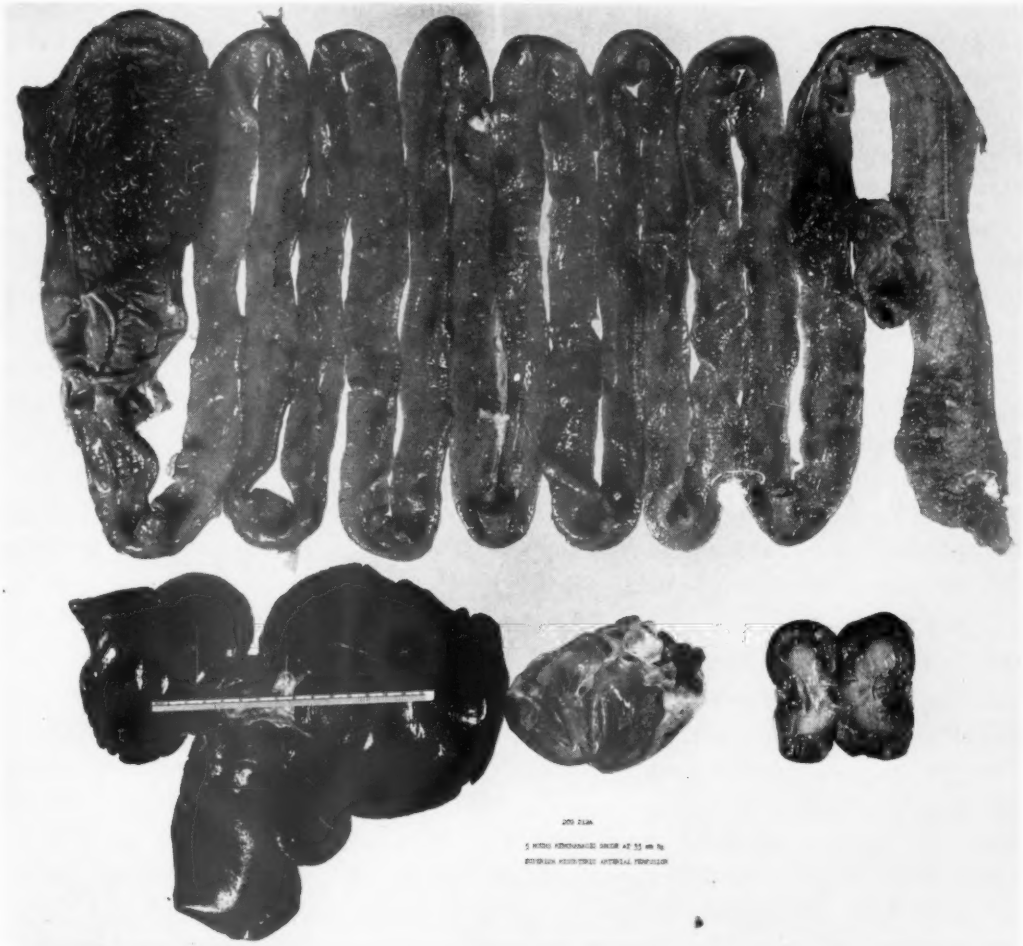

Fig. 5.—Viscera from a dog receiving superior mesenteric arterial perfusion during hemorrhagic shock. The dog was sacrificed a few hours following the experiment in order to inspect the various organs. The bowel was completely normal although the liver was markedly congested. The heart had a few subendothelial hemorrhages in the left ventricle. The kidneys appeared normal.

The majority of blood cultures taken from the blood reservoirs and from the dogs following retransfusion were sterile. When organisms were present there were fewer than 1,000 per ml. and they represented contaminants rather than invading bacteria from the bowel of the shocked dog. There was no difference in bacteriologic findings between survivors and nonsurvivors.

Because of the accuracy of the Sigmamotor pump it was not necessary to give the donor dogs any extra blood during the experiment. Moreover, blood volume studies done before hemorrhage and following retransfusion showed no significant blood volume changes in perfused dogs (Table II). There was no donor mortality due to irreversible shock after any of these experiments.

DISCUSSION

Several investigators have reported that irreversibility could be prevented by perfusing the livers of shocked dogs with arterial blood.[1, 2, 8, 10] All such experiments, with the exception of those of Frank and associates (1946),[8] have utilized anesthetized dogs. Moreover these investigators have started perfusion coincident with hemorrhage and have used a considerably shorter period of shock, usually 2 to 2½ hours, than was used in the present studies. By not using anesthesia during shock, we avoided the controversial VDM problem. The liver of anesthetized dogs in shock apparently produces VDM, a vasodepressor substance which is not produced by the liver of unanesthetized shocked dogs.[7] By initiating perfusion only after maximal bleeding volume was reached, neither the rate of bleed-out nor the ultimate maximal bleed-out volume was affected. Finally we used a 5-hour period of hemorrhagic shock to produce irreversibility since we found that almost all healthy unanesthetized or anesthetized dogs would survive 2 hours of hemorrhagic shock at 35 mm. Hg. These differences in technique make comparisons of results difficult. Nevertheless, the concept that the bowel is more susceptible than the liver to the ischemia occurring during prolonged shock is supported by recent studies. Edwards and associates (1954)[3] showed in dogs that occlusion of the aorta above the superior mesenteric artery but below the celiac axis for 2 hours resulted in a higher mortality than occlusion for a similar period just below the superior mesenteric artery with simultaneous hepatic artery occlusion. Parkins and co-workers (1955)[15] reported that differential intestinal cooling in dogs during a 2-hour occlusion of the thoracic aorta prevented death whereas differential cooling of the liver did not. Heretofore, the congestion seen in the bowel of dogs dying of irreversible hemorrhagic shock has generally been attributed to hepatic vasoconstriction with resultant portal congestion. However, we have demonstrated, as have Frank and his associates (1951),[6] that nonperfused chronic, Eck fistula dogs are not more resistant to irreversible hemorrhagic shock than normal dogs. Moreover, we found that these dogs also had mucosal congestion and necrosis in the bowel at autopsy despite the decompressing effect of the portacaval shunt.

The marked mucosal necrosis as well as congestion which is present in the bowel of dogs with irreversible shock has not been fully appreciated by many investigators. This necrosis is apparently the result of critical ischemia occurring during the shock period, possibly enhanced by the rich bacterial flora present. Superior mesenteric arterial perfusion during hemorrhagic shock

forestalled these changes and prevented irreversibility from occurring. Moreover, the survival of chronic Eck fistula dogs similarly perfused (Group II) ruled out increased portal flow to the liver as the basis for survival.

It is noteworthy that death in a shocklike state has been produced in dogs without hemorrhage by Erlanger and Gasser (1919)[5] and Penner and Bernheim (1939)[16] by the chronic injection of adrenalin. The only significant lesion present in these dogs at autopsy was mucosal congestion and necrosis of the bowel identical to that found in the bowel of dogs dying of irreversible hemorrhagic shock. It is probable that bowel ischemia resulting from the chronic adrenalin injections was also the cause of death of these dogs.

Recently, there have been several reports of the beneficial effects of pretreating experimental animals prior to hemorrhagic shock with adrenolytics,[12, 18] vasodilators,[9, 11, 20] or hypothermia.[14, 17] The salutary results obtained by these procedures have most likely resulted from increasing the blood flow to the bowel or from reducing oxygen requirements of the bowel during shock, thus preventing the development of lethal bowel ischemia.

Brain perfusion during hemorrhagic shock produced a definite increase in the duration of survival following retransfusion yet no significant increase in the number of permanent survivors over liver, aortic, or vena caval perfusion. Brain ischemia occurring during shock is evidently an important but sublethal factor in the genesis of irreversibility, because in hormotensive dogs the production of complete ischemia of the bowel by occlusion of the superior mesenteric artery leads to death only after 18 to 36 hours.[19] The precipitous death of dogs following irreversible hemorrhagic shock may be the result of the sublethal brain ischemia acting synergistically with the effects of lethal bowel ischemia.

SUMMARY

Irreversibility was prevented in 90 per cent of unanesthetized normal and chronic Eck fistula dogs by perfusing the bowel with arterial blood from a donor during a 5-hour period of hemorrhagic shock at 35 mm. Hg mean arterial pressure. Irreversibility was not prevented by perfusing the liver or systemic arterial or venous systems of unanesthetized dogs similarly shocked. Brain perfusion during hemorrhagic shock increased the duration of survival of the shocked dogs but did not prevent irreversibility from ultimately occurring.

I wish to thank Dr. Robert W. Clarke, Chief of the Department of Surgical Physiology, for encouragement and guidance during the course of these studies. Miss Anna Marie Link, Mr. Robert Kempf, and Mr. Jay Loquasto tirelessly assisted in the performance of these experiments. Dr. Joseph Preston of the Department of Experimental Biology prepared the tissue sections and aided in their interpretation. Mr. Charles McInturf of the Department of Experimental Surgery kindly made the bacteriologic studies.

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THE USE OF BLOOD TRANSFUSIONS IN THE TREATMENT OF THE POSTGASTRECTOMY SYNDROME

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SEVERAL theories have been advanced to explain the pathogenesis of the vasomotor symptoms which postgastrectomy patients with the dumping syndrome experience in the postprandial period. The most recent theory, stemming from the studies of Roberts and her associates,[1] postulates that these symptoms are initiated by an acute fall in blood volume, which occurs secondary to passage of a large amount of fluid into the gastrointestinal tract when hypertonic liquids or food are ingested.

Experiments performed in our laboratory tend to confirm this hypothesis, but we have encountered patients who experienced typical postprandial symptoms, with typical electrocardiographic changes, in whom the changes in blood volume did not exceed those observed in asymptomatic control patients. However, some of these patients had total blood volumes which were lower than the estimated normal, and it was postulated that under such circumstances a small change in blood volume might be sufficient to produce symptoms. It was further postulated that restoration of blood volume to normal levels by transfusion would cause an improvement in symptomatology, despite the fact that the postprandial fall in blood volume would probably be greater. We are reporting 2 patients treated in this manner.

CASE REPORTS

CASE 1.—A 27-year-old white man was first admitted to the Veterans Administration Hospital in November, 1952, with a long history of gastroduodenal ulceration complicated by hemorrhage, which had led to the performance of a subtotal gastrectomy in 1947. He remained asymptomatic for only a few months when ulcer symptoms returned and hematemesis recurred. A vagotomy was performed in 1949 following which time he remained asymptomatic for 12 months, when ulcer symptoms again recurred. He was then treated nonoperatively for marginal ulceration without improvement and was admitted to this hospital for further management on Nov. 11, 1952. Physical examination revealed a thin, pale, chronically ill white man who exhibited tenderness in the epigastric region of the abdomen. The usual laboratory examinations were within normal limits. A gastric analysis, using insulin as the stimulus for gastric secretion, revealed no free hydrochloric acid and a total acidity of 7 meq. per liter. A roentgenographic study of the upper gastrointestinal tract after ingestion of barium confirmed the diagnosis of marginal ulceration, and on Jan. 15, 1953, a second gastric resection was performed, excising the previous gastrojejunostomy including the ulcer, leaving an estimated 10 to 15 per cent of the stomach intact. Gastrointestinal continuity was re-established with an end-to-end jejunojejunostomy and an end-to-side gastrojejunostomy.

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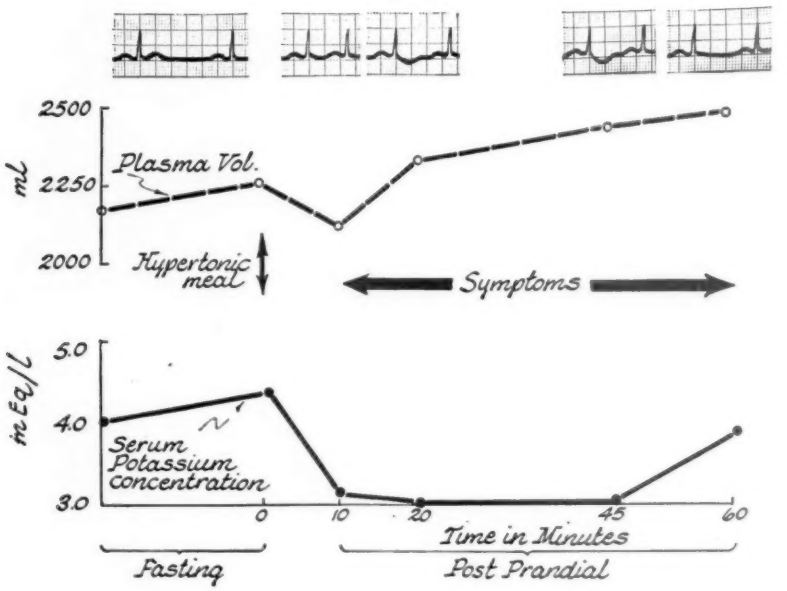
He did well for approximately 3 months when he developed symptoms typical of the dumping syndrome. These symptoms occurred while eating or within 10 to 15 minutes after the ingestion of a meal and consisted of nausea with frequent vomiting and diarrhea. There was an associated weakness, pallor, elevation of the pulse rate, and profuse sweating. His

Fig. 1.



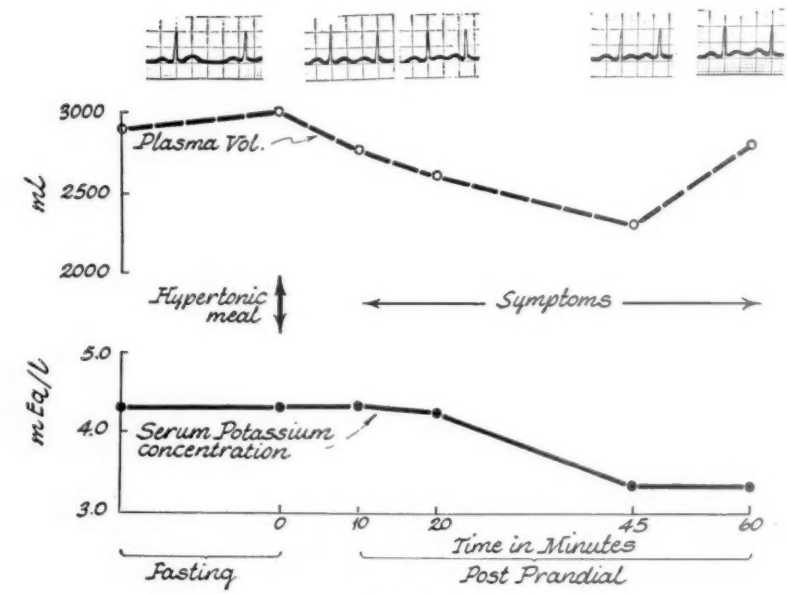


Fig. 2.

Fig. 1.—A diagram illustrating the alterations in the electrocardiographic tracing, plasma volume, and serum potassium concentration after ingestion of a hypertonic meal prior to transfusion therapy. The patient experienced severe symptoms.

Fig. 2.—A diagram illustrating the alterations in the electrocardiographic tracing, plasma volume, and serum potassium concentration after ingestion of a hypertonic meal after transfusion therapy. The patient experienced very mild symptoms.

symptoms would often last as long as 2 hours, during which time he was so weak that he could scarcely lift his head from the pillow. Physical examination showed no particular changes since his previous admission.

Tests demonstrated that symptoms occurred following ingestion of carbohydrate, protein, or fat. A study of jejunal motility exhibited extreme hypermotility during the symptomatic period and anticholinergic drugs failed to decrease postprandial bowel motility. Jejunal aspiration studies revealed no evidence of afferent loop obstruction. A psychiatric consultation was obtained, and it was the opinion of the psychiatrist that none of the patient's symptoms were on a psychiatric basis.

Many different medical regimens were tried during a 4-month period, most of which time the patient remained in the hospital. These included alterations in the diet, at one time progressing to a complete protein diet, recumbency after meals, and anticholinergic drugs. Tincture of belladonna, phenobarbital, hexamethonium, Banthine, Dactil, and Piptal were administered singly and in combination in addition to variations in the diet with no relief of symptoms.

On July 27, 1954, a jejunoplasty was performed, designed to create a jejunal pouch at the site of anastomosis. It was thought that this procedure might increase his effective gastric capacity somewhat and that the pouch would allow some mixing of bile and pancreatic juice with food before it proceeded further into the jejunum. During this procedure, he was given 500 ml. of blood.

His postoperative course was uneventful and his postgastrectomy symptoms improved to the point that he was able to return to work, although they were not completely relieved. Before surgery he had had symptoms following each meal. In the immediate postoperative period, he experienced symptoms only 2 to 3 times a week, but during the subsequent 18 months the symptoms gradually increased in severity until January, 1956, when they were as severe as they had been at any time. Further attempts at medical management with alterations in diet and use of antispasmodics were made without relief of symptoms.

The patient was then readmitted to the hospital on April 9, 1956, for additional studies. Cholangiography was within normal limits. Roentgenographic examination of the upper gastrointestinal tract after ingestion of barium showed rapid motility of the bowel with complete emptying of the stomach within 15 minutes. The head of the barium column was in the transverse colon within this period of time. Serial plasma volume determinations in the postprandial period, using T-1824 dye, following the ingestion of a hypertonic liquid meal demonstrated a fall in plasma volume of only 96 ml., but the patient experienced severe symptoms and an electrocardiographic tracing made simultaneously with the blood volume determination revealed marked changes with an increase in pulse rate, the disappearance of the T wave, and the appearance of a U wave. Serum potassium determinations performed simultaneously revealed a fall in serum potassium of 1.30 meq. per liter. On the basis of the patient's weight at the time of this admission, his estimated normal blood volume was 5,100 ml., whereas his actual calculated blood volume was 3,676 ml. On the basis of his normal weight prior to the onset of his present illness, his estimated normal blood volume was 6,800 ml. Consequently it was decided to make a therapeutic test, administering Dextran rapidly while the patient ingested a hypertonic liquid meal, which always in the past had produced symptoms. When this was performed, he was able to ingest the meal without developing symptoms. Therapy was then instituted using whole blood, and a total of 2,000 ml. of blood was transfused, 500 ml. daily for 4 days. On May 13, 1956, 3 days after completion of the transfusion therapy, the measured blood volume was 5,814 ml. Ingestion of the hypertonic liquid meal at this time produced a fall in plasma volume of 350 ml. but the patient experienced only minimal symptoms. Electrocardiographic alterations were again noted but were less marked than previously (Figs. 1 and 2). He was discharged from the hospital on May 19, 1956, with instructions to remain on his diet. The patient has been followed at periodic intervals since that time and was last seen in January, 1957, at which time he was asymptomatic and working regularly.

CASE 2.—A 39-year-old white man was admitted to Hermann Hospital, on July 3, 1954, with symptoms suggesting acute perforation of a duodenal ulcer. His previous health had been good with the exception of typical ulcer symptoms since 1945, intermittently relieved by alkalies and diet. Laparotomy was performed 10 hours after the onset of symptoms. A free perforation of an anterior duodenal ulcer was found, and an immediate 80 per cent gastric resection was performed reconstituting gastrointestinal continuity with a gastroduodenostomy. His postoperative course was uneventful and he was discharged from the hospital on the tenth postoperative day, taking a 6-feeding bland postgastrectomy diet. His weight at the time of discharge was 154 pounds. He was seen at monthly intervals for 3 months postoperatively, at which time his weight was 152 pounds and he was eating a completely unrestricted diet without discomfort of any kind.

The patient was next seen 18 months after surgery, and at that time his weight was 130 pounds. He appeared nervous and apprehensive and gave a classic story of symptoms of the dumping syndrome of approximately 3 months' duration, consisting of weakness and palpitation occurring approximately 15 minutes after his meals, usually more severe following lunch. His symptoms would subside within a short time if he was able to lie down. Physical examination was not remarkable except for the obvious weight loss, nervousness, and apprehension. The patient was instructed to take a minimal amount of liquids with his meals, to lie down for 15 minutes after eating, and to avoid foods with a high carbohydrate content, and phenobarbital was prescribed one-quarter grain to be taken 4 times daily. When seen again one month later, he had had only one symptomatic episode. The patient stated that the phenobarbital made him too sleepy; therefore, Serpasil was prescribed in a dose of 0.25 mg. twice daily.

The patient did not return for follow-up visits until July 7, 1956—2 years postoperative—at which time his weight was 123 pounds and he was again complaining of severe dumping symptoms, generalized fatigue, and frequent palpitation. Again, physical examination revealed no specific abnormalities other than tachycardia of 100 beats per minute, obvious lassitude, and weight loss.

An upper gastrointestinal series revealed a normally functioning gastroduodenal stoma without evidence of ulceration, but gastric emptying was rapid. Liver function studies and peripheral blood studies were within the limits of normal; however, a blood volume determination using T-1824 dye revealed a plasma volume of 2,038 ml. and a total blood volume of 3,774 ml. This represented a deficit of 1,000 ml. on the basis of his weight at that time and a 2,000 ml. deficit on the basis of his ideal weight. The patient was placed on a high vitamin, high caloric diet, and over a period of one week was transfused with 1,500 ml. of whole blood. There was a complete and prompt disappearance of all symptoms of the dumping syndrome.

He remained well until Sept. 26, 1956, at which time he noted lassitude, developed a low-grade fever, and became jaundiced. He was again hospitalized and liver function studies were compatible with a diagnosis of serum hepatitis. He remained in the hospital until Oct. 21, 1956, and showed continuous clinical and laboratory improvement. He has taken a high protein, high caloric, high vitamin diet since the diagnosis of hepatitis was made without distress of any kind. When last seen in January, 1957, he remained asymptomatic and his weight had slowly returned to 131 pounds.

DISCUSSION

The proper management of patients with symptoms following subtotal gastrectomy is predicated upon an accurate appraisal of the etiologic factors involved, for different pathophysiologic alterations may produce symptoms, which, on casual examination, appear similar. The syndrome exhibited by the 2 patients reported herein has commonly been referred to as the dumping

syndrome. In our experience 35 to 45 per cent of all patients treated by subtotal gastrectomy experience symptoms of this nature to some degree. Most of them have a normal blood volume, and fortunately symptoms are usually mild, so that the patient requires no medical assistance in alleviating his distress. Of those patients who need medical advice, the majority are satisfactorily managed by the elimination of offending foods, ingestion of a dry, low carbohydrate diet, and, when indicated, the prescription of antispasmodic drugs and sedatives. Recumbency after meals may also be beneficial. The number of patients who fail to respond to this regimen is small, but each one constitutes an extremely difficult therapeutic problem. Several operative procedures have been advocated for the treatment of this syndrome, but none has given uniformly good results. One such procedure was utilized in Case No. 1 and seemed to produce partial, temporary relief. In retrospect it seems possible that the blood which was administered may have been a factor in his improvement.

It is impossible to make unequivocal statements based upon an experience with only 2 patients, but the fact that the clinical response in each correlates well with objective physiologic measurements suggests that the transfusions produced relief of symptoms. Therefore, since blood volume determinations and blood transfusions can be accomplished easily, this therapeutic approach seems justified in all patients with protracted postgastrectomy symptoms.

SUMMARY

Physiologic studies of postgastrectomy patients with the dumping syndrome indicate that a postprandial fall in blood volume initiates the vasomotor symptoms. Patients who have a normal blood volume will tolerate such changes more readily than those in whom the blood volume is low. Consequently, when symptoms are difficult to control, a blood volume determination is indicated, and if the blood volume is found to be low, restoration to normal levels may aid in the control of symptoms. Two illustrative cases are reported.

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MODIFIED SUCTION APPARATUS

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A METAL suction tip with the necessary rubber tubing connection is needed during most operative procedures. Being attached to a distant suction source, these arrangements are fairly standard. The type of suction tip used will depend upon the operation being performed and the preference of the operating surgeon.

The constant noise of the suction during the operation can be most irritating. Generally, occlusion of the rubber tubing is the only means of stopping this noise.

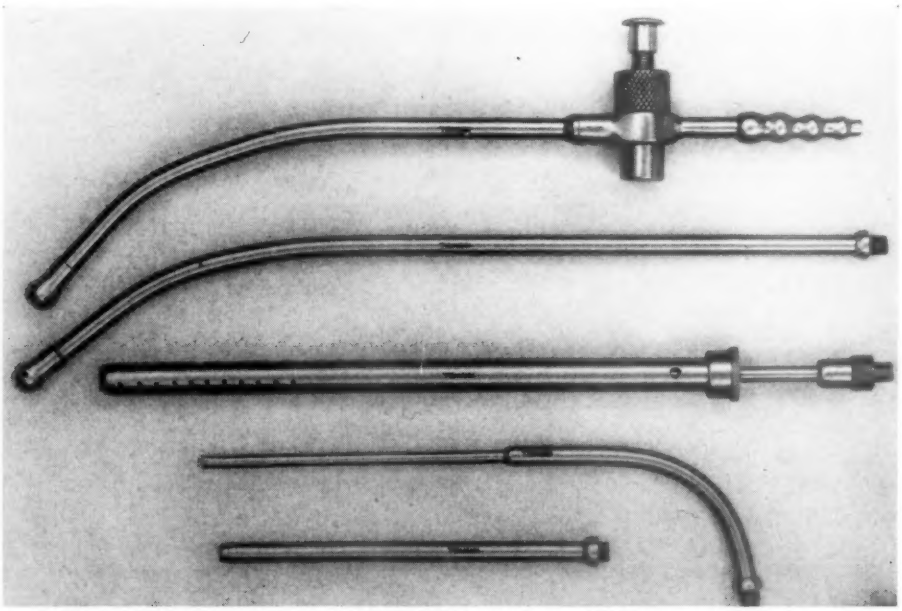

Fig. 1.—The main unit with the shut-off valve is shown at the top with the Yankauer tip attached to it. From above downward are 4 other interchangeable suction tips: (a) thoracic tube (Yankauer type), (b) Pool, (c) Elsberg, and (d) plastic surgery type.

The suction tip† pictured here (Fig. 1) has been designed to eliminate the noise as much as possible and still allow the use of several conventional suction tips. The main portion of the unit has a spring valve which is easily operated by the thumb. When it is pressed there is both suction and noise. When it is released there is neither suction nor noise. The spring valve unit is easily removed, if necessary, for cleaning purposes. The screw threads on the main unit and on the ends of the various interchangeable suction tips make this a versatile suction for all types of surgery.

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†Can be obtained from the manufacturer: George Tieman & Co., 107 East 28th Street, New York 16, New York.

THE IMPEDANCE PLETHYSMOGRAPH

Its Clinical Application

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A GREAT variety of instruments has been devised for the study of peripheral hemodynamics and the evaluation of local circulatory changes in various experimental and disease states. Basically, most of these instruments record or measure some changes in physical characteristics, such as volume, pressure, temperature, and even transmission of light occurring in a portion of the body. From these physical changes, various physiologic phenomena, such as blood flow, blood pressure, and vasomotor activity, can be estimated with varying degrees of accuracy. Considering the number of variable physical and physiologic factors involved in the study of hemodynamics, this diversity of instrumentation is not surprising. It is doubtful that any one instrument short of an analogue computer will ever permit the proper integration of all the factors involved.

The impedance plethysmograph, as its name suggests, was designed to record changes in the volume of body segements,[4, 6, 7] but it functions on an entirely different principle than that of the conventional volume displacement instruments. The biophysical background and the principles on which this instrument are based have been exhaustively reviewed by Nyboer.[6] It is an electronic instrument which graphically records and measures minute variations in the impedance of a body segment to a radio-frequency current in the neighborhood of 150 to 200 kilocycles. Basic research at the time the instrument was proposed indicated that body tissue behaved as a homogeneous conductor of electrical current at that frequency, much the same as a piece of wire acts as the conductor of direct current, and, as such, the ability of a digit or an extremity to conduct that current would be related to its length and cross-sectional area. It was then postulated that since the product of length and cross-sectional area is equal to volume, and conductivity is a reciprocal function of impedance or resistance, changes in impedance should be mathematically related to changes in volume. Graphic recordings of impedance variations in various body segments, when inverted to correspond to changes

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in conductivity, very closely resemble volumetric plethysmograms obtained simultaneously from the same segments.[3-7, 13] The mathematical relationship between the volume and the impedance or conductivity of the body segment which was formulated by early investigators,[4, 6, 7] however, was not substantiated in venous occlusion experiments.[5, 13] Consequently, the use of the impedance plethysmograph as an instrument for studying blood volume and blood flow has been limited by the lack of a standard of reference. It, therefore, has not been possible to convert units of electrical measurement into units of volume. Furthermore, recent studies[11] indicate that body tissue at the frequencies employed is not a homogeneous conductor of electrical energy.

Despite these theoretical and experimental objections to its use as a volume recorder, the impedance plethysmograph, by design, is a sensitive electronic instrument which measures and records minute variations in electrical conductivity of body segments. In this respect it is similar to instruments used for the conductometric analysis of electrolyte solutions.[12] In these studies, the solution whose electrical properties are to be studied is placed in a container called a conductivity cell. This cell incorporates two electrodes between which an alternating current of a given frequency is passed. In impedance plethysmography two electrodes are applied to a body segment and an alternating current is passed between them. What happens to the electrical current as it passes through the electrolyte solution in the conductivity cell or in the body segment can be recorded or measured in many different ways, but the principles are the same. The analogy between the conductivity cell and the body segment is apparent and is important to a clear understanding of the principles involved in impedance plethysmography in contrast to other forms of plethysmography.

The purpose of this paper is to show that the impedance plethysmograph is of value in the study of peripheral vascular conditions by virtue of its basic function as a recorder of certain electrical properties of body segments. The factors involved in evaluating its usefulness in clinical practice are not unlike those encountered in electrocardiography and electroencephalography. These instruments also record certain electrical phenomena, the exact source and nature of which are not yet completely explained, but their empiric use has proved of great value to the clinician. Furthermore, it is important to note that it has not been considered necessary or even desirable to relate the electrical measurements to any other physical parameter, such as pressure, volume, or temperature, in order to interpret the information derived from their use.

MATERIAL AND METHODS

The material to be presented was selected from our experimental and clinical experience with the impedance plethysmograph. Several experimental studies are included to demonstrate the principles involved in this type of instrumentation. In addition, a few representative studies on patients with peripheral vascular disease are presented to show some of its clinical applications.

The instrument used by us was constructed in accordance with the circuit of DuBois and Nims.[4] The electrodes consisted of fine silver bands shown in the insert in Fig. 1. Four bands were used in the experimental studies on

fingers.[8] The Sanborn Twin-Viso ECG unit was used as the end recorder. No integrating or differentiating circuits were employed.[6] The polarity of the signal fed from the impedance plethysmograph proper to the end recorder was reversed so that a positive deflection of the tracing represented a decrease in impedance (or an increase in conductivity) and vice versa.

The tracing obtained with this instrument was calibrated by comparing its amplitude to the deflection made by the addition of a known resistance. This was done while another resistance equal to the resistance of the segment was substituted for the segment. In this way, the pulsations did not interfere with the injected standard. Knowing the value of the standard resistance, the height of the deflection it causes, and the height of the pulse wave, the amplitude of the pulse wave can be determined in terms of ohms. For the sake of simplicity, however, we have omitted all calculations. The standardizing deflections which appear on the tracings show up as two heavy lines due to the

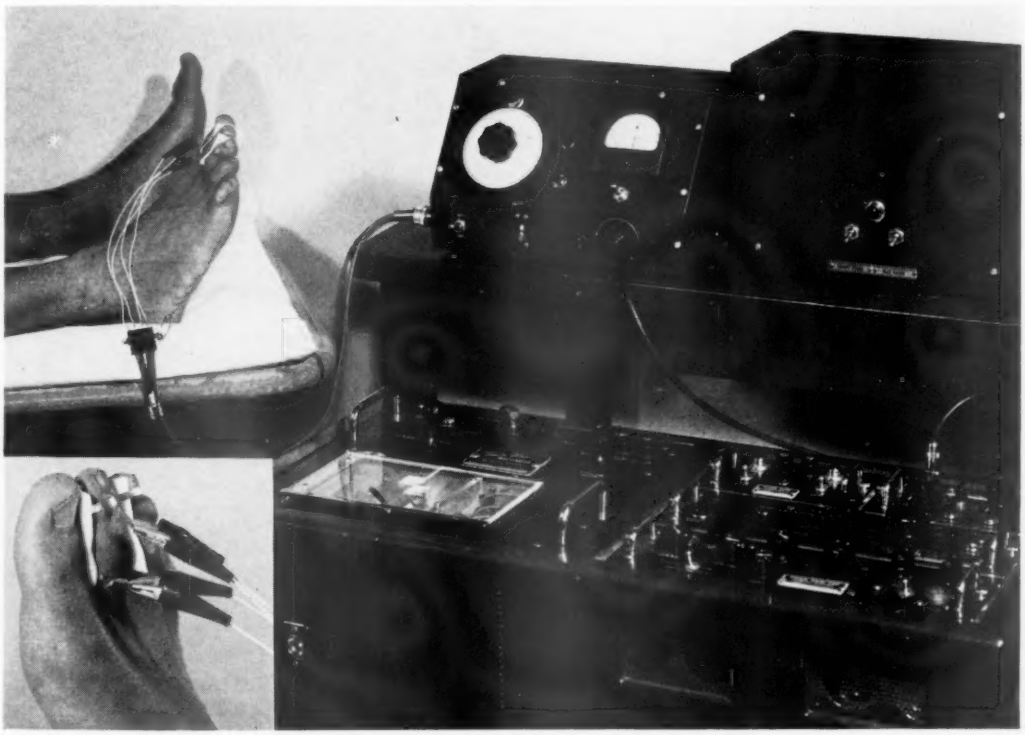

Fig. 1.—Photograph of an impedance plethysmograph with its power supply. The recorder is in the foreground. The insert is a close-up of the electrodes and leads in position.

rapid excursion of the stylus. They are shown without calculations as an indication of the relative sensitivity or amplification. So long as the sensitivity or amplification does not change, increases or decreases in the recorded pulse amplitude are immediately obvious and can be monitored on an oscilloscope and recorded on paper as desired.

Although individual pulse waves are recorded quite accurately, the base line or position of the tracing on the paper does not always exactly follow the

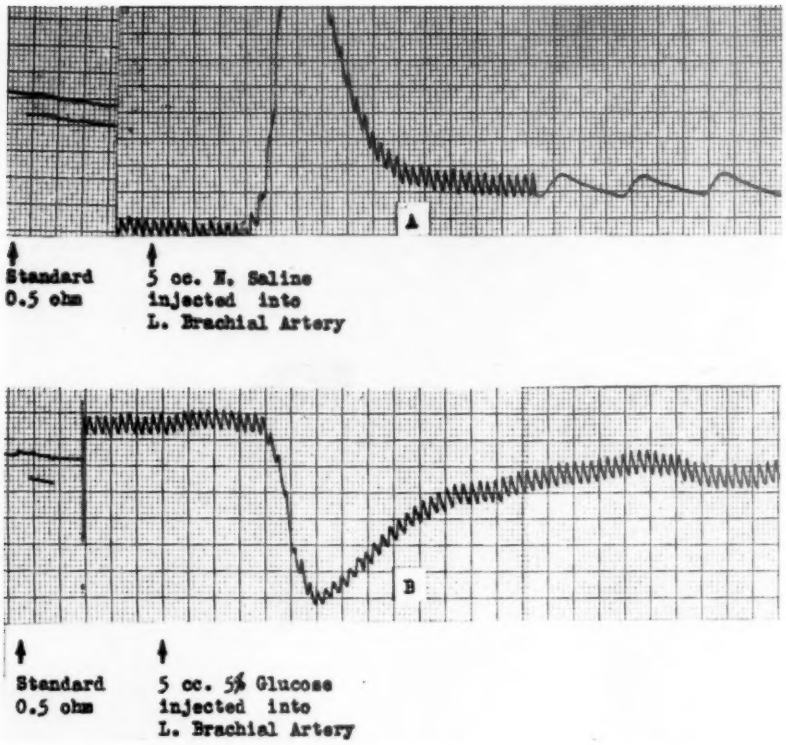

Fig. 2.—*A*, An impedance plethysmogram of the left index finger, 5 c.c. of normal saline having been injected into the brachial artery. (The chart speed was increased for the last three pulsations shown.) *B*, An impedance plethysmogram of the same finger, 5 c.c. of 5 per cent glucose having been injected into the brachial artery proximally.

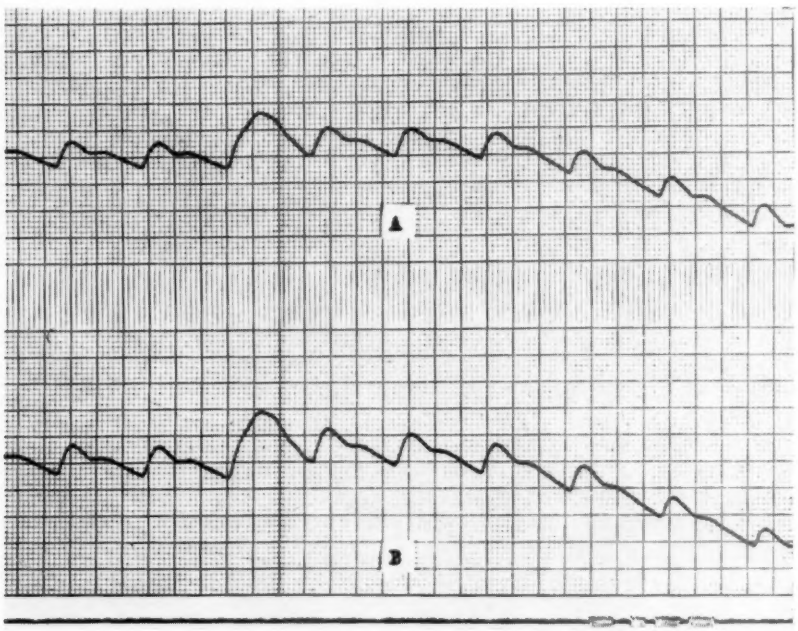

Fig. 3.—*A*, Impedance plethysmogram of a segment of finger. *B*, Mechanical plethysmogram of the same segment recorded simultaneously. The marker at the bottom signaled that the patient coughed toward the end of the tracing.

gross changes in the over-all impedance of the segment when the ECG preamplifier is employed. The decay characteristics of this preamplifier cause the stylus to drift back to its original setting. This must be accepted in order to gain increased amplification of the pulse wave and still keep the tracing on the narrow paper chart. The degree of amplification is ultimately limited by the effects of hum, electronic noise, and especially motion or tremor on the part of the patient.

RESULTS

The tracings shown in Fig. 2 were recorded without using the preamplifier. The marked shifts in the base lines of the pulse waves, therefore, display accurately the changes in the over-all electrical conductivity of the finger. When the injected sodium chloride, a better conductor than whole blood, reached the finger, there was a sudden increase in conductivity. When the same volume of glucose, a poorer conductor than blood, reached the finger, there was a decrease in conductivity. These tracings suggest that the impedance plethysmograph responds to changes in ionic concentration within the vascular compartment. There has been no evidence to assign a vasodilating effect to normal saline nor a vasoconstricting effect to 5 per cent glucose. This experiment strongly suggests that the body segment becomes a conductivity cell and that the changes in conductivity as recorded are mediated for the most part by the vascular compartment.

Further evidence of this fact can be noted in Fig. 3. The lower tracing (*B*) was made by enclosing the finger in a plastic cylinder. Changes in pressure in the air surrounding the finger were recorded by means of a statham strain gauge and a strain gauge amplifier. The electrodes of the impedance plethysmograph were placed directly under the seals at each end of the cylinder and recorded the electrical changes occurring in the same volume of finger. Again, without using the preamplifier, both tracings appear virtually identical. It should not be inferred that any mathematical relationship necessarily exists between these two wave phenomena because the techniques are probably measuring different physical parameters and the amplitude of the impedance recording was reduced so that it was approximately equal to that of the strain gauge recording. Nevertheless, if it is accepted that the strain gauge recorded changes in the finger mediated by the vascular compartment, then it seems reasonable to conclude that the impedance plethysmograph recorded changes, for the most part, from the same source.

Fig. 4 demonstrates the increase in pulse amplitude in a normal toe occasioned by homolateral paravertebral block and recorded by the impedance plethysmograph. The setup for this and subsequent examinations is the same as depicted in Fig. 1. Two silver bands were applied; one limited proximally by the web space, and the other limited distally by the eponychium. By observing the height of the standardization deflection prior to each run, it can be seen that the instrument sensitivity or degree of electronic amplification has

not changed appreciably, and yet there has been a large increase in the amplitude of the recorded wave. Since the recorded phenomena are similar to those obtained by conventional plethysmography and sphygmography, we feel that the changes shown in Fig. 4 are representative.

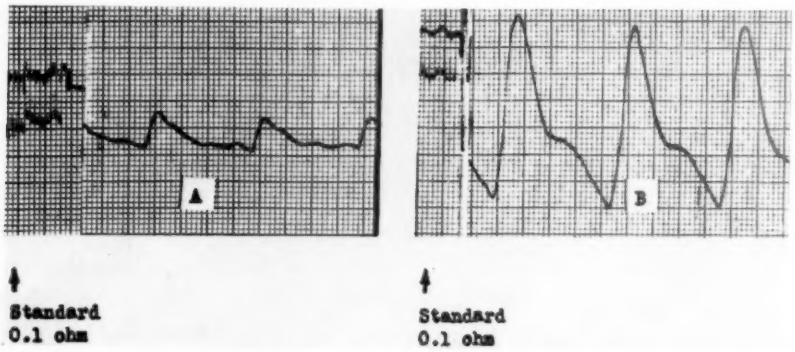

Fig. 4.—*A*, Impedance plethysmogram of a segment of normal second toe after equilibration to an ambient temperature of 20° C. *B*, Same segment following homolateral paravertebral block.

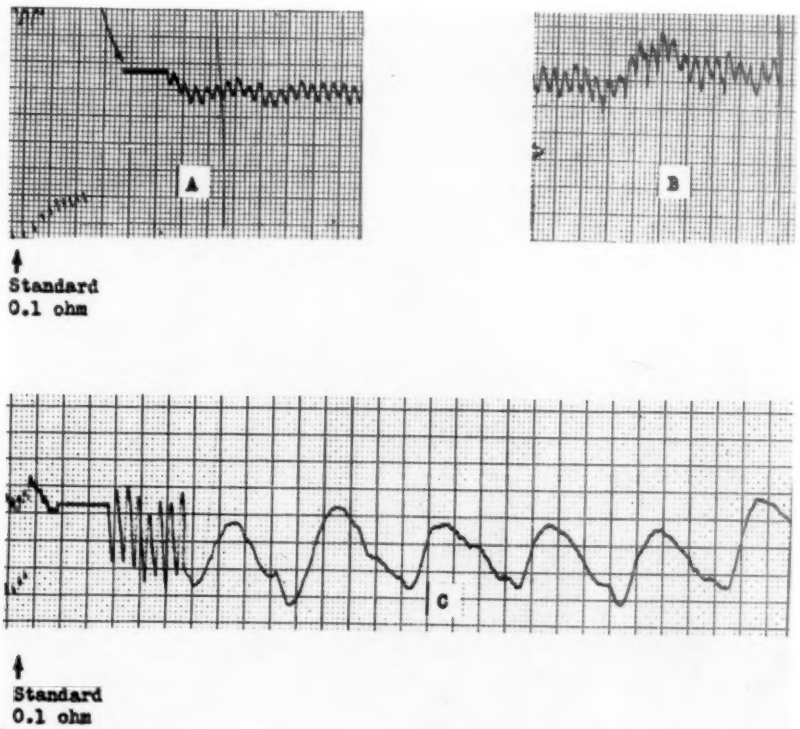

Fig. 5.—*A*, Impedance plethysmogram of a segment of second toe of a patient with moderate obliterative disease. *B*, Same, after paravertebral block. *C*, Same segment of toe one week following lumbar sympathectomy. The chart speed is increased during most of the tracing.

Although norms have not been established, it is possible to have the subject or patient serve as his own control. The electrodes can be replaced on the same toe with reasonable accuracy by using anatomic landmarks or artificial

skin marks. The tracings shown in Fig. 5 were obtained from the toe of a patient with moderate obliterative vascular disease, characterized by absent pedal pulses and paresthesias of the great toe. A slight increase in pulse amplitude is seen after paravertebral block, and an even greater response is noted after sympathectomy. Note that the instrument sensitivity has been almost cut in half in the postoperative tracing, so that the increase in pulse amplitude was even greater than it appears.

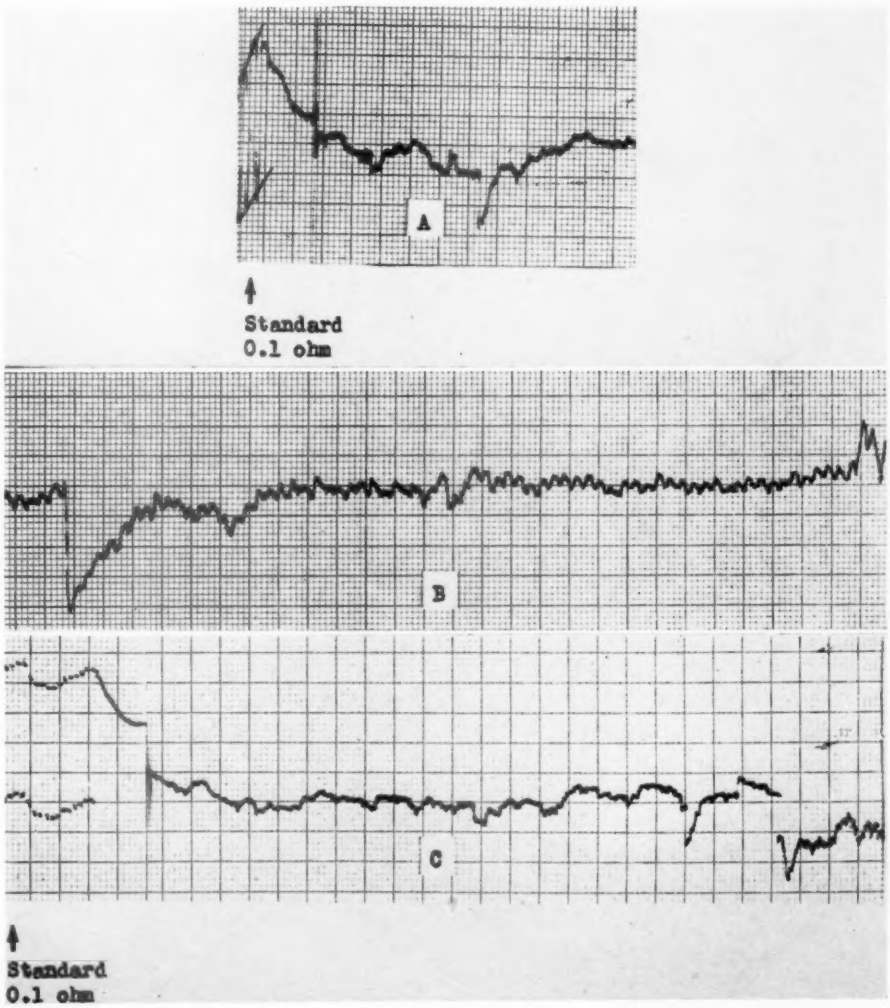

Fig. 6.—*A*, Impedance plethysmogram of a segment of second toe of a patient with severe obliterative disease. No pulse wave visible. *B*, Same toe after paravertebral block. *C*, Same toe one week following popliteal graft and lumbar sympathectomy. Again, no organized pulse wave is detectable.

In contrast, the patient whose tracings appear in Fig. 6 suffered from severe intermittent claudication. An arteriogram revealed a block involving the length of the popliteal artery and no visible filling below the distal third of the lower leg. The disorganized deflections due to noise and motion in (*A*) acquired some semblance of pulsation after paravertebral block. A tracing

(not shown here) after sympathectomy failed to show a pulse wave, as did tracing (*C*) made after the insertion of a nylon prosthesis. This patient subsequently developed gangrene of the foot and required a leg amputation below the distal end of the prosthesis.

Another interesting application of this instrument is shown in Fig. 7. With the recent advances in the use of arterial grafts and prostheses, there has been an increased interest in the exact physiologic significance of pulsatile blood flow.[9, 10] Tracings (*A*) and (*B*), obtained from the regions directly over the popliteal prosthesis on one side and the popliteal aneurysm on the other side, were made simply by attaching the electrodes to a plastic bar and affixing this to the skin over the palpable pulsation at symmetrical points on each leg. Without more experimental and empiric information in this field of study, no conclusions can be drawn at this time as to the significance of the wave configurations.

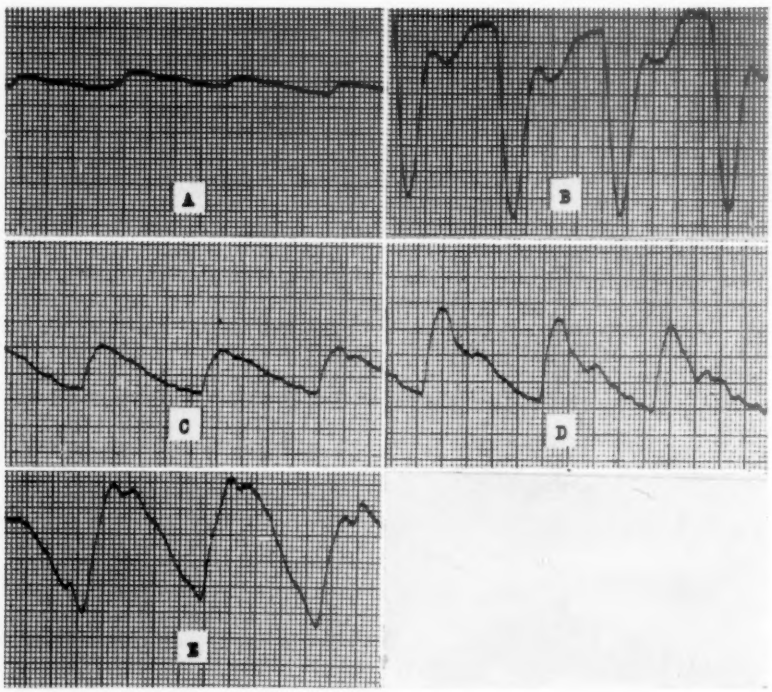

Fig. 7.—*A*, Impedance plethysmogram made by placing electrodes on the skin over a nylon popliteal graft. *B*, Plethysmogram made by placing the same electrodes over an unoperated popliteal aneurysm of the other leg. (Sensitivity the same as in *A*.) *C*, Impedance plethysmogram of second toe distal to the nylon graft. *D*, Impedance plethysmogram of second toe distal to the aneurysm. (Same sensitivity as in *C*.) *E*, Impedance plethysmogram of the index finger of the same patient. (No proximal aneurysm.)

DISCUSSION

Volumetric plethysmographs, despite infinite variations in transducers, amplifiers, and end recorders, all have one thing in common. They all require that the body segment studied be sealed in a rigid vessel containing the medium which transmits volume changes to the transducer. These vessels usually consist of plastic cups, tubes, or cylinders, and the accuracy of this type of plethysmography as a means of estimating blood flow and volume changes depends

on the care with which these devices are applied. Elimination of tissue distortion, temperature effects, avoidance of leaks, and correction for the accumulation of perspiration are some of the problems involved.[1, 2] Furthermore, if the results of the study of one body segment are to be compared with those of another or the same one at a later date, the absolute volume of each segment must be accurately measured.[1]

In clinical practice, the greatest demand for plethysmography is in evaluating the effects of nerve block, ganglionectomy, vasodilator drugs, and arterial grafts or prostheses on the circulation of diseased lower extremities, more specifically, the toes. These toes tend to exhibit all manner of deformity, variation in size and shape, and are often easily traumatized. For these reasons, the general application of venous occlusion techniques in plethysmography is not practical. Because of the difficulty in measuring absolute volumes of the toes, the application of conventional plethysmography in this area is restricted to studies in which the toe serves as its own control. The results are usually interpreted on the basis of qualitative changes in the amplitude or shape of the pulse waves observed over a short period of time.

If the shortcomings of conventional plethysmography in clinical practice are recognized, then impedance plethysmography deserves a trial on its own merits. It is a very sensitive detector of pulsatile phenomena in small body segments. The body segment is connected to the instrument by means of electrodes which can be adapted to fit any surface or appendage and are applied as easily as thermocouples. They can be reapplied to the same area with more accuracy and with greater facility than appliances used in conventional plethysmography. Thus, long-term comparisons on the same patient are simple and reliable.

We have constructed three units based on the circuit of DuBois and Nims.[4] The basic circuit is satisfactory, but the preliminary adjustments (tuning) would be greatly facilitated and the over-all sensitivity improved if transformers specifically engineered for the circuit could be obtained. Once the instrument is properly tuned and calibrated, however, it is remarkably stable and will operate from a fairly well-regulated power supply. Its accuracy and proper functioning can always be checked, simply by switching in a calibrated resistor in place of the part to be studied. It contains no mechanoelectric transducer, the characteristics of which might influence the final wave form or require correction if the wave form were subjected to analysis. The chosen frequency of 175 kilocycles, although theoretical objections have been raised against it,[11] is not a difficult frequency to work with. It is worth speculating that with proper engineering and design this instrument could be made to function completely by frequency modulation, rather than by a combination of amplitude and phase modulation. In that event, the effects of stray pickup and hum which are so prevalent in a hospital environment might be largely eliminated.

SUMMARY AND CONCLUSIONS

Since 1943, when Nyboer, Nims, and Bagno[7] submitted their circuit for an impedance plethysmograph to the National Research Council, any clinical

potentialities which the instrument might have had were eclipsed by theoretical and experimental objections to original claims made for it. It would have been preferable had it not been called a plethysmograph or volume recorder, because it is a device which measures electrical conductivity. Conductivity, a reciprocal function of resistivity or impedance, is a distinct and measurable parameter of any conductor. The problem of whether conductivity is quantitatively related to tissue volume, blood flow, or other properties of living tissue becomes an academic matter if the information derived from its empiric measurement is of value in its own right and in its own absolute units of measurement.

When compared to conventional plethysmography, impedance plethysmography appears to yield equally valuable information for the clinical evaluation of circulatory changes in the toes.

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Case Reports

CARCINOMA OF THE LUNG DEVELOPING IN A CONGENITAL CYST

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WOMACK and Graham,[5, 6] in 1941, were probably the first to suggest that congenital cysts of the lung predispose to the development of carcinoma. There have been, however, few well-documented cases in which a patient with known cystic disease of the lung was followed over a period of years and then developed a carcinoma in the wall of the cyst.

In 1947, Moersch and Clagett[4] reported 2 patients, 22 and 36 years of age, respectively, who were operated upon for removal of a lung cyst and were found to have an associated carcinoma. Korol[2] reported 10 cases of carcinoma in middle-aged war veterans with known cystic disease of the lung. Seven of the tumors developed in a group of 40 patients who had been followed for 13 years. He stated that the incidence of carcinoma in 500 cases of cystic disease of the lung accumulated from the literature was 9 per cent, while the incidence of pulmonary cancer in necropsied adults was approximately 1.5 per cent.

In 1954, Anderson and Pierce[1] recorded 6 cases of carcinoma of the bronchus presenting as thin-walled cysts. Although they had no record of a pre-existing cyst in any of these patients, they suggested that: "This variety of bronchial carcinoma may arise when a thin layer of malignant cells grows into and lines a pre-existing cavity." Another patient with a cyst of the lung and an associated carcinoma was reported by Larkin and Phillips,[3] in 1955.

This report is concerned with a patient who had a cyst of the lung for at least 16 years before she developed any symptoms. At operation she was found to have a carcinoma which appeared to have developed in the wall of the cyst.

CASE REPORT

A 34-year-old woman (M. H.) was admitted to the hospital complaining of fever, chest pain, and a cough of about 2 months' duration. She stated that at the age of 17 years she had a chest roentgenogram as part of a routine physical examination and was told that she had a cyst in her right lung. No treatment was advised. Roentgenograms at the age of 25 years and 29 years (Fig. 1) showed a thin-walled cyst, presumably unchanged in size, and without evidence of fluid. Roentgenograms on admission to the hospital showed a cavity in the right upper lobe which contained fluid (Fig. 2). It was thought that the patient had an infected cyst, and operation was advised.

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On opening the chest, the right upper lobe of the lung was found to be densely adherent to the chest wall necessitating extrapleural dissection to free the lung. The lobe was then removed.

Pathology.—Gross: The specimen was a right upper lung lobe weighing 280 Gm. The apical pleural surface over an area 7 cm. in diameter was shaggy and firm. The remainder of the pleural surface was smooth and shiny. A stiff-walled cavity (Fig. 3), 10 cm. in diameter, was located in the upper portion of the lobe and partially filled with semiclotted blood. The lining surface was tan and rough. The cyst wall varied from 0.1 to 4.0 cm. in thickness. The thickest portion was firm, grayish pink, and had a lobular, discrete periphery suggesting tumor emanating from the cyst wall. A 4 cm., firm, smooth surfaced, and bosselated mass (Fig. 4), in continuity with underlying tumor, projected into the cavity lumen in an area near the hilum of the specimen. Bronchi were displaced by the cavity with the exception of one which directly entered the wall of the cyst and was occluded by tumor. Pulmonary arteries and bronchi, except as noted, were otherwise unremarkable.

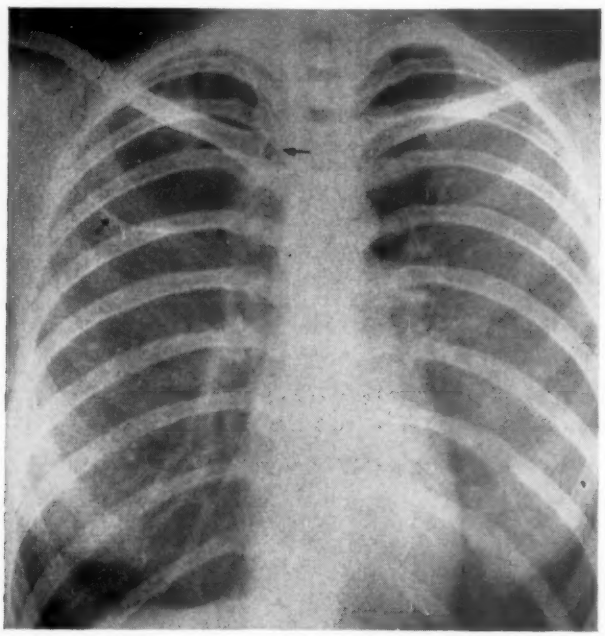


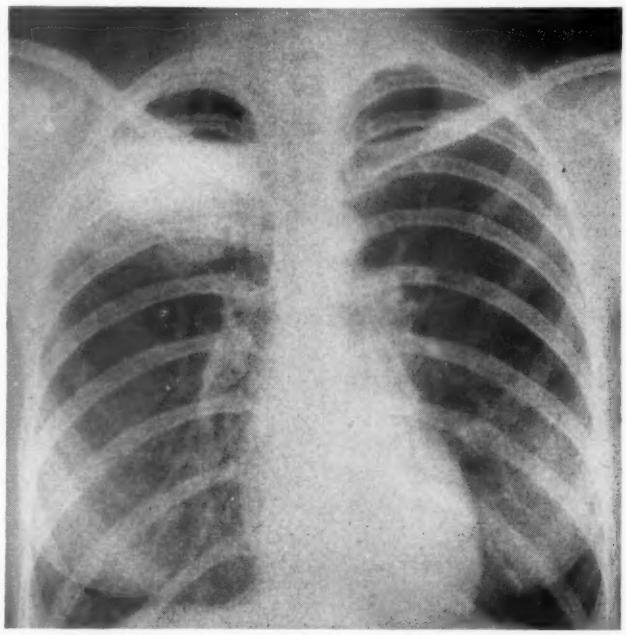
Fig. 1.—Roentgenogram of chest 5 years before operation. Patient's age, 29 years.

Microscopic: The majority of the cyst wall was composed of a very undifferentiated tumor with numerous areas of acute necrosis. Tumor cells (Fig. 5) were predominantly large with abundant granular eosinophilic cytoplasm in varying shapes and lacked cohesiveness. Nuclei were large, lobulated, and hyperchromatic with prominent nucleoli. Bizarre giant cells (Fig. 6) with multiple nuclei were conspicuous as well as "strap cells" suggestive of rhabdomyoblasts. Special stains failed to reveal cross striations in the cytoplasm. In a few areas, tumor cells were in ill-defined clusters (Fig. 7). Mitoses were not infrequent. The histologic appearance of the tumor cells was interpreted as indicating epithelial origin. The fibrillary stroma was diffusely infiltrated by polymorphonuclear leukocytes and lymphocytes. The surrounding lung parenchyma showed partial atelectasis and many macrophages as well as irregular lumina lined by hypertrophied terminal bronchiolar cells. The latter lumina were separated by interstitial extension of tumor.

The cyst lacked a specific type of lining cell or other structures to identify its exact nature or origin. Fibrin was deposited on the inner surface. The apical or thinnest portion of the cyst wall was dense fibrohyaline tissue with irregular spaces lined by terminal bronchiolar epithelium.

Diagnosis.—Pulmonary cyst associated with undifferentiated carcinoma of the righ upper lung was the diagnosis.

The patient's immediate postoperative course was uncomplicated, but she soon developed evidence of recurrent and metastatic disease and died about 3 months after operation.



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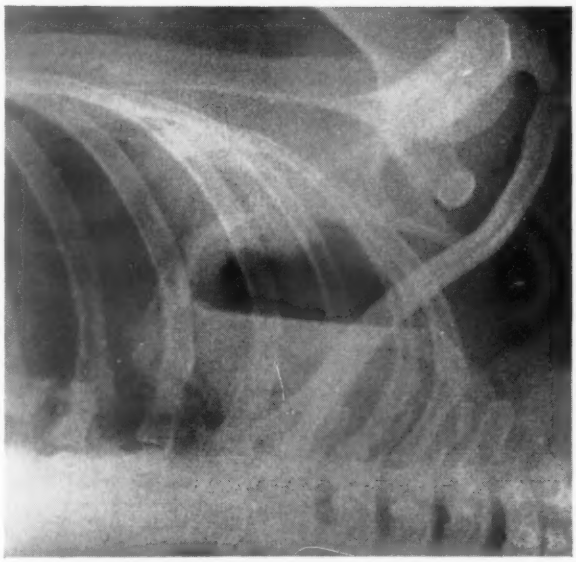


B.

Fig. 2.—*A,* Roentgenogram on admission to hospital. Patient's age, 34 years. *B,* Shows fluid level with patient in lateral decubitus position. Irregular shadow on lateral wall is probably caused by tumor.

A necropsy revealed histologically similar tumor invading the thoracic wall as well as metastases to mediastinal and mesenteric lymph nodes, gastrointestinal tract, and liver. There was no evidence of other pulmonary cysts in the left lung or right lower lung, nor evidence of chronic or congenital bronchial disease.

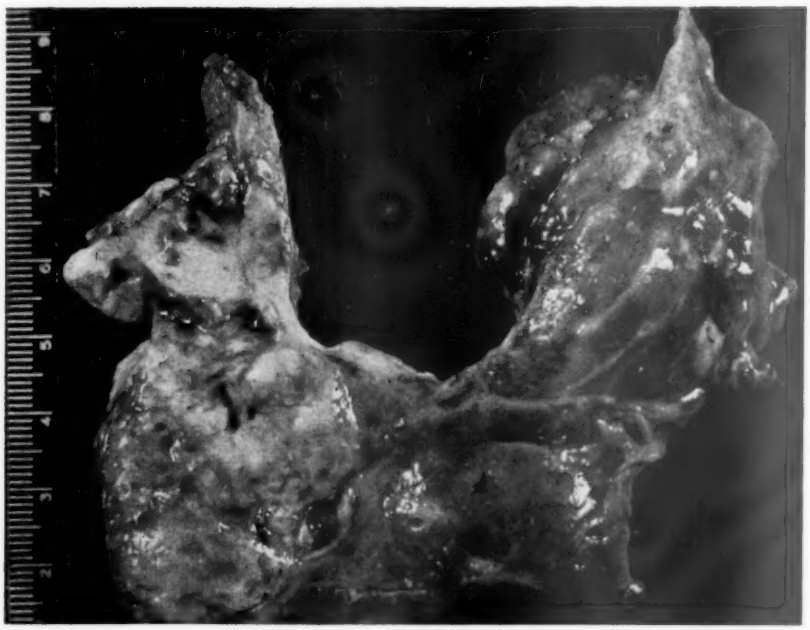
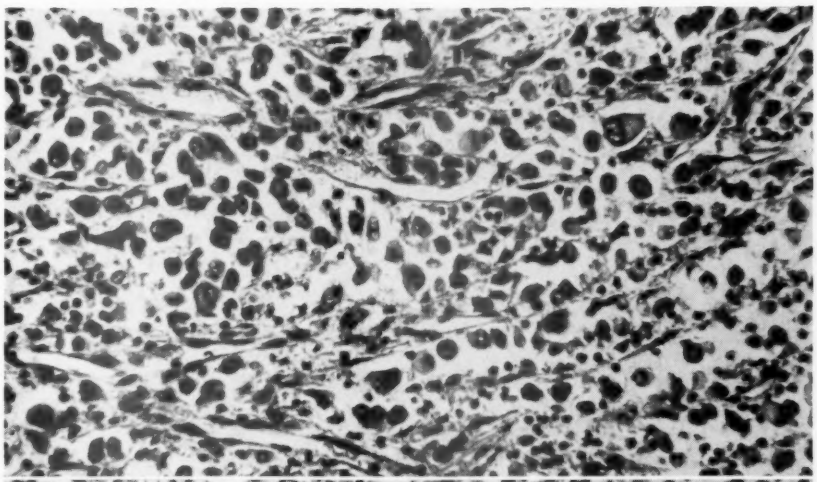


Fig. 3.—Representative cross section of cyst showing tumor emanating from the wall projecting into lumen.

Fig. 4.—Cyst opened to show the fibrinous lining and intraluminal extension of a bosselated tumor mass.



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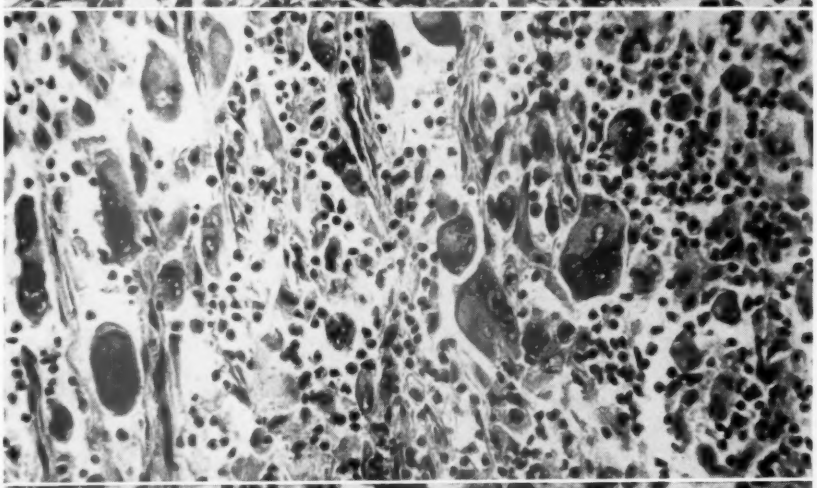

Fig. 6.

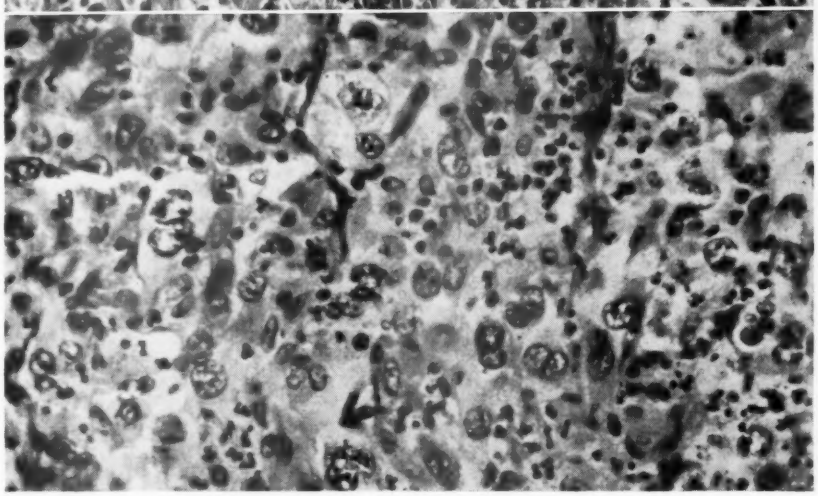

Fig. 7.

Fig. 5.—Loosely arranged undifferentiated tumor cells in a fibrillary stroma infiltrated by leukocytes. (Hematoxylin and eosin; ×170.)

Fig. 6.—Bizarre giant tumor cells with multiple hyperchromatic nuclei. (Hematoxylin and eosin; ×170.)

Fig. 7.—Clusters of tumor cells suggesting the epithelial nature of the tumor. (Hematoxylin and eosin; ×170.)

SUMMARY

A 34-year-old woman, known to have a pulmonary cyst for at least 16 years, was operated upon for removal of the cyst and found to have an associated carcinoma. It is believed that the cancer developed in the cyst wall.

A review of the available literature indicates that congenital cysts of the lung are potentially malignant and therefore should be excised.

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OBSTRUCTION OF THE COMMON BILE DUCT DUE TO ASCARIS LUMBRICOIDES

Report of a Case Following Subtotal Gastrectomy

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THE migratory characteristics of the *Ascaris lumbricoides* (roundworm) within the human body are well known. The rather common affinity of this parasite for the biliary system has been commented upon by Butt[1] who reviewed necropsies and reported an operated case involving the gall bladder. Karlen[2] also reviewed autopsy reports of this condition, dealing primarily with cases in foreign countries. Keller[3] points out that the condition is more common than realized in the United States. Reported cases of surgical significance in reference to the common bile duct, however, are comparatively rare in the United States and Great Britain. A case is discussed in the *British Medical Journal* by Smyth[4] and a case in the United States has recently been reported by Shore.[5]

The following is a case report of common duct obstruction by roundworms following a subtotal gastrectomy in a patient who had a choledochoduodenal fistula, apparently due to penetration of a posterior duodenal ulcer.

CASE REPORT

H. M., a 44-year-old white farmer, was admitted to the surgical service of the North Carolina Baptist Hospital in September, 1955, complaining of abdominal pain, primarily epigastric.

Present Illness.—For eight years prior to the present admission the patient had noted dull, inconstant aching pain in the epigastrium accompanied by occasional nausea and vomiting, which was infrequent and unrelated to meals. For the last 2 years his difficulty had increased with more frequent and more intense epigastric pain. Occasional colicky pain with radiation around the right costal margin had been present for 2 months. Food and milk would relieve symptoms temporarily. A barium study of the stomach and duodenum was done elsewhere, revealing a duodenal ulcer. Medical therapy was advised and instituted. One month prior to admission the symptoms became much worse. A repeat barium study revealed a deformed duodenal bulb with barium visualized in the common and hepatic ducts. There had been no history of diarrhea, melena, acholic stools, or jaundice.

Past History.—Intermittent constipation with prolapse of hemorrhoids for the past 7 years. No history of intestinal parasites elicted.

Physical Examination.—Abnormal physical findings were confined to the abdomen which was generally tender on palpation, more so in the epigastrium and right upper quadrant. Internal and external hemorrhoids were present to a moderate degree.

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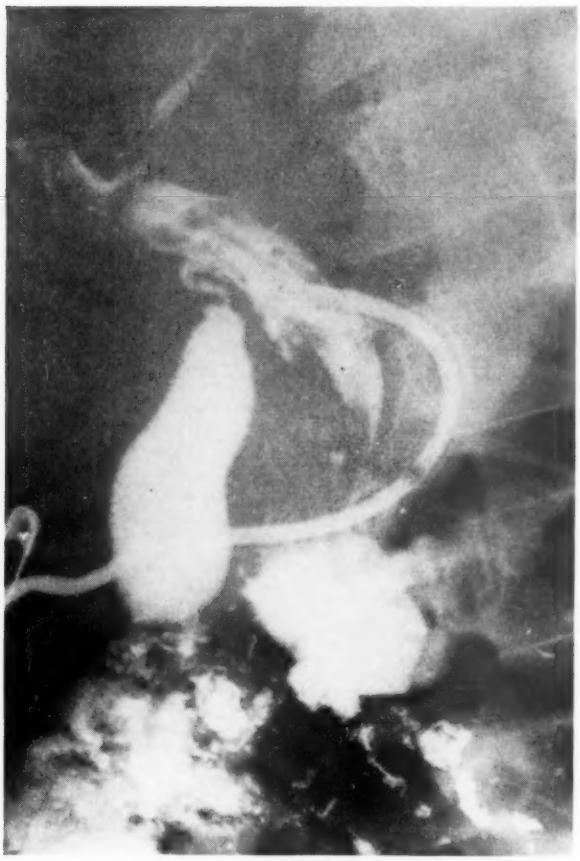

Fig. 1.—Cholangiogram revealing roundworms in the common and hepatic bile ducts.

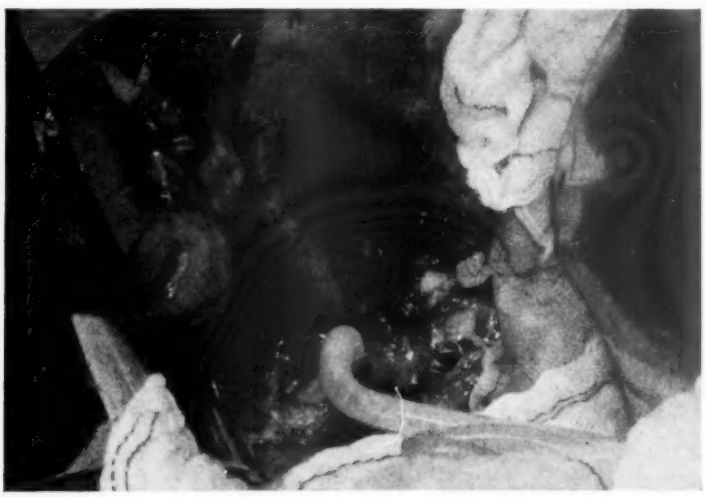

Fig. 2.—Roundworm emerging from opening in common bile duct during second operation.

Accessory Clinical Findings.—Complete blood count and urinalysis were within normal limits. No eosinophils were seen on differential. Fasting gastric analysis showed 22 units of free acid.

Roentgenogram.—Oral and intravenous cholangiography demonstrated a functioning gall gladder without evidence of stones. Barium study of the stomach and duodenum revealed partial obstruction of the duodenum with marked pyloroduodenal deformity and gas and barium in the biliary tree.

Hospital Course.—On the basis of the x-ray evidence of duodenal deformity with partial obstruction and a choledochoduodenal fistula, surgical exploration was carried out on Sept. 22, 1955, under general anesthesia. The gall bladder was normal in appearance, contained no stones, and emptied readily. The first portion of the duodenum was markedly scarred and partially obstructed. Exploration of the remainder of the abdominal viscera demonstrated numerous *Ascaris lumbricoides* in the jejunum and ileum. The duodenum was opened revealing a 3 cm. posterior wall ulcer in the base of which was located a fistulous tract communicating with the terminal portion of the common bile duct. This tract was doubly ligated and divided. Approximately two-thirds of the stomach, in addition to the first portion of the duodenum was removed, the duodenal stump closed and an antecolic Polya gastrojejunostomy accomplished. A T tube was placed in the common duct, brought out through a lateral stab wound, and the incision closed in layers.

Wangensteen suction via Levin tube was instituted immediately and discontinued on the third postoperative day. On the fourth postoperative night the patient complained of increasing epigastric paid radiating to his back. The following morning he was noted to be jaundiced. Serum bilirubin was 7.0 mg. per cent. His abdomen had become rigid with rebound tenderness. A T tube cholangiogram showed round worms obstructing the common duct (Fig. 1). At surgery that same afternoon, 5 *Ascaris lumbricoides* were removed from the common duct (Fig. 2). The parasites in the stomach and jejunum were "milked" to the mid-jejunum and 6 of them removed from a small incision in the bowel. Those remaining in the ileum were expressed into the cecum where 5 were removed through the base of the appendix following its excision. Ten centimeters of piperazine citrate was injected into the jejunum and 10 cm. into the cecum. A T tube was reinserted in the common duct and an operative cholangiogram was done, showing good filling of the biliary tree with no obstruction and no further worms in the common or hepatic ducts. As soon as solid food could be tolerated by the patient, a course of oral piperazine citrate was given. Cholangiogram prior to discharge was normal. Stool examination was negative for ova or parasites. He was discharged 18 days following his second operation with the T tube in place. One month later, during his follow-up visit, a normal cholangiogram was obtained and the T tube removed from the common duct. He has continued to do well.

COMMENT

Ascaris lumbricoides are cosmopolitan in distribution and are common in poorly sanitated areas. In the United States they are endemic primarily in the southeastern parts of the Appalachian range where 30 to 40 per cent of the population has been found to be infected.

In man, the parasite resides in the small intestine, subsisting on partially digested food and perhaps intestinal villi. The ova are passed in the feces, mature in the soil, and are ingested via contaminated food, drink, and dirt. The larvae pass into intestinal capillaries, enter the portal circulation or the thoracic duct, and are filtered out eventually by the lung. From the lung they migrate up the respiratory passages to the epiglottis and are swallowed, reaching maturity in the small intestines. Normally they reside in the lower jejunum and ileum where they are truly parasitic.[6]

When the food supply of these parasites is withheld from the human alimentary tract their propensity to migrate leads them to other areas in search of nourishment. Invasion of the biliary and pancreatic ducts with obstruction, breakdown of anastomotic sites by perforation, and entrance into the stomach with subsequent regurgitation and aspiration may occur.

In endemic areas, the surgeon should maintain a high index of suspicion and institute vermifuge therapy prior to elective surgery in patients found to have ova in the stools. If *Ascaris lumbricoides* are encountered during elective or emergency surgical procedures following which the patient's oral intake must be deferred, their surgical removal seems advisable in order to prevent the potentially serious postoperative complications.

As noted in the case report, several roundworms were discovered to be distributed throughout the small intestine during the initial surgical procedure. To avoid further surgery at that time they were not removed, since institution of vermifuge therapy was planned as soon as the patient could tolerate solid food. Apparently the search for nourishment led 5 of these parasites into the common bile duct, causing subsequent obstruction.

SUMMARY

1. A report of obstruction of the common bile duct by *Ascaris lumbricoides* (roundworms) following subtotal gastrectomy is presented.

2. Reported cases of surgical significance involving the common bile duct are relatively rare in the United States.

3. *Ascaris lumbricoides* found in the intestine during a surgical procedure in which oral feeding must be deferred should be removed, lest serious postoperative complications result.

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APPENDICOURETHRAL FISTULA CAUSED BY A FOREIGN BODY

A Case Report

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APPENDICOURETHRAL fistula caused by ingestion of a foreign body is an extremely rare entity not previously reported.

Higgins,[2] in 1936, tabulated 441 cases of vesicointestinal fistula of which 3 per cent involved the appendix. In a similar study of 592 cases, Kellogg[4] noted 27 instances (4 per cent) of appendicovesico fistula. In both of these series the fistulas were almost entirely secondary to appendicitis associated with subsequent abscess formation.

Most authors[1] have reported an incidence of 2 to 3 per cent of foreign bodies in cases of acute appendicitis. Similarly, Mahoney[7] has listed 74 cases in which a foreign body was felt to be the cause of the appendicitis. In 40 of these, pins were the offending objects.

A careful search of the medical literature reveals only 4 cases of appendicovesico fistula due to foreign bodies with a pattern similar to that of the present case.[3, 5, 6, 8] In each of these there was a history of ingestion of a pin with subsequent stone formation in the bladder and eventual fecaluria and pneumaturia.

CASE REPORT

O. L., a 25-year-old man, was first seen in the Columbia-Presbyterian Medical Center in 1944. At that time he gave a history of having had a straight pin and two calculi removed from his bladder at the age of 13 in Santo Domingo. He brought his roentgenograms with him (Fig. 1). The patient had no knowledge of how the pin got into the bladder. A further history was obtained of a cystotomy for removal of a calculus at 17 years of age, and a nephrolithotomy 4 years later.

He returned to this hospital in 1945, at which time a periurethal abscess required incision and drainage. Two years later, he reappeared with complaints of severe pain and burning on urination. An intravenous pyelogram was negative. The symptoms gradually subsided.

In 1948 the patient came in with a story of having passed tomato seeds in his urine. For the first time, two chronically draining sinuses were evident in the area between the scrotum and the rectum. A diagnosis of the rectourethal fistula was made despite failure to demonstrate the tract on proctoscopy, cystoscopy, and repeated urethrograms. A diverting colostomy was advised, but refused.

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He was next seen in 1953 requesting operation for a persistent fecaluria and dysuria. A cystogram at this time outlined a tract between the bladder and the cecum (Fig. 2).

Abdominal exploration was carried out on Feb. 25, 1953. A thickened, indurated, but benign-appearing appendix was found. The distal tip of the appendix was definitely fused to a cordlike tract running along the back of the bladder to the posterior

Fig. 1.

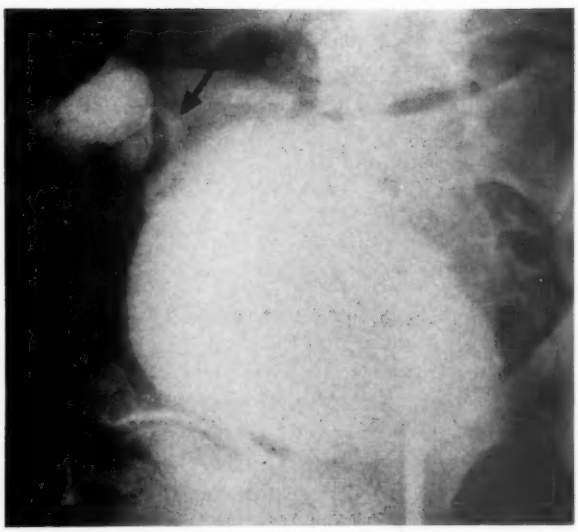

Fig. 2.

urethra. The fistulous tract was divided and ligated at its entry into the bladder, and an appendectomy was also performed. The pathologic specimen showed a chronically inflamed appendix in continuity with a fistulous tract.

Postoperatively, the patient has done well to date except for a single brief episode of pyelonephritis and periodic urethral dilatations for control of an uretheral stricture.

SUMMARY

A case of appendicourethral fistula caused by a foreign body, which posed a difficult diagnostic problem, and to the best of our knowledge, is the first of its kind, has been presented.

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Recent Advances in Surgery

CONDUCTED BY ALFRED BLALOCK, M.D.

MALIGNANT MELANOMA OF THE FEET AND HANDS

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REAPPRAISAL of the results of the treatment of malignant melanoma has long been a prime consideration of the Mixed Tumor Service of the Memorial Cancer Center. We have been fully cognizant of the obligations imposed by an abundance of material which in recent years matches in number of yearly admissions that of one of the most common malignant tumors of the body, gastric cancer. The effect of the site or location of the melanoma, the mode of behavior, the choice of treatment, and the end results of therapy are the most important and definitive considerations if the over-all experience is sufficiently extensive.

There are no 2 homologous organs in the body which present more remarkable variation in neoplastic diseases as the hands and feet and yet present such minor variation in structure. A consideration of the tumors of these sites begets numerous speculations into the causes of these variations. The beginning of this change in these homologous organs, the feet and the hands, has been indicated by Wolff[60] who has stated that "The signal change in human evolution was due to the upright gait, and when man lifted his hands from the ground they were free from the purpose of locomotion and put into the service of intelligence."

It is difficult to assign specific causes for such variations in these respects and any statements are open to much speculation as to what variant factors were brought to bear upon these 2 organs once the upright posture was assumed, leaving the foot predominantly as an organ of locomotion and the hand for the function of prehension. One can understand the difference of incidence of epithelial tumors, especially skin cancers and keratoses which have been shown to be subject to certain environmental factors and exposure. Therefore, their frequency on the hands and difference in gross characteristics from those developing in the skin of unexposed surfaces of the body is anticipated. Yet it is difficult to explain the incidence and distribution of nevi on these 2 sites, with the predominance of blue nevi on the hands as opposed to their infrequency on the feet, likewise the preponderant incidence of junctional neval change on the feet, where it is 6 times as frequent as on the skin surface in general and twice that of the hands.

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A second reason for this analysis is that it is part of a more comprehensive report on a regional consideration of tumors. Tumors of the hands and feet do not constitute a large group in most reports. In a series of 757,827 persons registered in The Henry Ford Hospitals and Clinics, Clifford and Kelly[20] found an incidence of 9,741 (1.3 per cent) pathologically verified malignant tumors. In this group, 63 (0.65 per cent) were primary tumors of the hands. This number represents only 0.008 per cent of the entire registered hospital and clinic population. This contrasts with a 20-year survey by Schreiner and Wehr[52] at the Roswell Park Institute during which time 128 malignant tumors (1.1 per cent) of the hand were found in a series of 11,212 malignant neoplasms. At about the same time, a 19-year survey of 10,459 cases of malignant tumors revealed that 37 (0.35 per cent) arose on the feet.[53] The latter figures indicate a higher than usual occurrence rate of malignant tumors because they come from an institute specializing in neoplastic diseases. The opinion has been generally held that benign tumors greatly preponderate over malignant neoplasms of the hands and feet. For instance, in a recent review from the surgical pathologic laboratory of one of our large metropolitan teaching hospitals presented before the American Society for Surgery of the Hand, 1,203 benign tumors were reported in contrast to 143 which were malignant.[55] Mason,[35] reviewing 700 tumors of the hand, found 12 per cent to be malignant. Our experience has not duplicated this preponderance of benign tumors, probably because there is a more selected material coming to the Memorial Cancer Center. In a previous survey of a 20-year experience (1917 to 1938) with tumors of the hands and feet on the Mixed Tumor Service at the Memorial Cancer Center, Pack[43] found a total of 204 benign and 369 malignant tumors of the hands. In another study which overlaps the preceding analysis in a 13-year period from 1935 through 1947, we have found 347 benign and 196 malignant tumors of the hands and 155 benign and 151 malignant tumors on the feet, a total of 502 which were benign and 345 malignant.[13]

Reports of strictly regional considerations of neoplasia effecting either the hands or feet are not numerous. Aside from studies mentioned, Mason[36] has presented an extensive report on tumors of the hands. A symposium on the subject was edited by Pack[44] for SURGERY in January, 1939. L. D. Howard,[31] John Howard,[30] and Caulfield[17] have issued recent reports. A brief series of articles on the subject considered the problem with reference to office surgery for the ambulatory patient.[63] However, a detailed account of neoplasia confined to the hands and feet has not been published.

A study of our case histories from the Memorial Cancer Center has indicated that the prevalent over-all impression of the benignity of tumors in these locations has generally contributed to failure in early detection and diagnosis and, not uncommonly, inadequate initial surgical consideration. Our experience has not been that of one authority on hand surgery who has stated that malignant tumors of the hand are rare, but, when they do occur "due to their peripheral location and early diagnosis, they afford a good chance for cure."[31] In the hope that this statement might come to represent a more universal experience, we should like to discuss the problem in detail, for it has been long since

this problem has been reconsidered in its manifold aspects. Contrariwise, we do not concur entirely with a recent introduction to this problem that "fortunately for man the occurrence of malignant tumors of the hand is rare. When they do occur, they are usually highly malignant and that is particularly true of the melanotic group."[17]

In a prior consideration of cancers of the hands and feet based upon our patients treated between 1935 through 1947, there were 196 malignant tumors of the hands and 151 on the feet. Squamous and epidermoid cancer was the most frequent tumor, with 72.4 per cent of the malignant tumors of the hands being in this group but with only 9.2 per cent of those on the feet. Second in frequency was malignant melanoma which reverses the picture, with a 9.7 per cent incidence on the hands and 59 per cent on the feet. Kaposi's disease was next in numerical importance on the feet; malignant synovioma was third in frequency on the hands.

The purpose of this paper is to assess the factors influencing treatment of melanoma of the hands and feet and to study the natural history of melanoma on these sites. These data are part of a continuing study which extends from 1935 through 1954 numbering 174 cases on the feet as opposed to 41 on the hands. Because of numerical preponderance, greater consideration will be given to malignant melanomas of the feet. Publication of these data is believed to be timely because of many conflicting viewpoints in appraisal of end results in the treatment of melanoma and especially to evaluate a large series of regional node dissections in the groin and axilla in the control of melanoma from specific sites and perhaps in this way to clarify the ambiguity that may arise from more composite reports in which the sites of origin of these melanomas have not been strictly considered.

In our experience the most common malignant tumor of the feet and the second most frequent malignant tumor of the hands and feet considered together is malignant melanoma. By definition melanoma or melanocarcinoma pertains only to malignant tumors of melanoblasts. At the Memorial Cancer Center the term is not used to denote any benign pigmented skin lesion; the exception, juvenile or prepubertal melanoma is accorded a modifying term implying its benignity, as well as its histologic simulation of the malignant lesion. There were 151 cases of malignant melanoma of the hands and feet during this 16-year period from 1935 through 1950, during which time 917 patients with melanoma of various parts of the body were examined and treated at the Memorial Cancer Center. Therefore, 16.5 per cent of the patients with melanoma during this period had it either on the hands or feet, whereas the total combined skin surface of these 2 areas is only 10.5 per cent.[11]

In a survey of the primary sites of melanoma in 851 cases reviewed by Pack, Perzik, and Scharnagel,[47] extending through the period of 1917 to 1946, a total of 13.7 per cent of the lesions were on the upper extremity, with 10.1 per cent located outside the hand and 2.3 per cent on the hands, with another 1.2 per cent located beneath the nails. In the same period, 27.0 per cent of

the melanomas were on the lower extremity, 12.7 per cent were found exclusive of those on the feet, 12.2 per cent occurred on the feet exclusive of those beneath the nails, and 2.2 per cent were in the subungual position. Pack, Gerber, and Scharnagel[46] in a later analysis of 1,190 cases extending from 1917 to 1950 regrouped their cases and considered the palm separate from the arm and the sole separate from the leg, because of the difference of epidermal density and the distinctive natural history of the disease in these locations. In this analysis, 1.3 per cent were found in the palm, 9 per cent were on the sole, and 2.9 per cent were subungual. Allen and Spitz[5] in a similar consideration of 934 cases found 11.3 per cent on the upper extremity, 0.6 per cent on the palms, and 1.9 per cent subungual, contrasting with 29.7 per cent of their series on the lower extremity of which 10.2 per cent of the entire series were on the soles and 1.8 per cent subungual. Stewart, Hay, and Varco[54] reported a 13 per cent incidence of melanomas on the toe and feet (with a total of 28 per cent on the lower extremity), 2.2 per cent on the finger, and 1 per cent subungual of 92 cases. These incidences of sites contrast Wright, Clark, and Milne's[62] report of 132 cases, of which 54 were on the lower extremity, with only 2 of them subungual and one involving the sole.

MELANOMA OF THE FEET

Melanoma occurs much more frequently on the feet than on the hands, although the skin surfaces of these areas are about the same. The greatest incidence is between 50 and 70 years of age with about the same number of patients in each of these decades (Table I). Women comprise 59.8 per cent of the group; men 40.2 per cent. Ninety-seven and five-tenths per cent were white, and 2.5 per cent were Negro.

TABLE I. MELANOMA OF THE FEET
Age, Sex, and Racial Distribution

	TOTAL		MALES		FEMALES	
AGE IN YEARS	NUMBER	PER CENT	NUMBER	PER CENT	NUMBER	PER CENT
Total	122	99.9	49	100.0	73	100.0
10 and less	—	—	—	—	—	—
11-20	1	0.8			1	1.3
21-30	6	4.9	2	4.1	4	5.5
31-40	20	16.4	7	14.3	13	17.8
41-50	23	18.8	9	18.4	14	19.2
51-60*	32	26.2*	11	22.4	21	28.8
61-70	30	24.6	13	26.5	17	23.3
71-80	10	8.2	7	14.3	3	4.1
Range: 12 years (Female) to 77 years (Male)						

SEX	NUMBER	PER CENT	RACE	NUMBER	PER CENT
Males	49	40.2	White	119	97.5
Females	73	59.8	Negro	3	2.5

*Greatest frequency.

Melanoma in the Negro.—In the entire group at the Memorial Cancer Center, Allen and Spitz[5] calculated on the basis of the ratios in the hospital

population that the incidence in white and Negro population was 3.7 to 1. It has been previously demonstrated that melanoma occurs in a statistically high proportion of Negro patients on the foot, and Baxter,[8] collecting 170 cases of melanomas in the colored races, found that 111 (65.3 per cent) occurred on the foot as opposed to only 16 (9.4 per cent) on the remaining part of the lower extremity and only 13 (7.6 per cent) on the entire upper extremity. Charache[18] reported the only incidence of malignant melanoma in a Negro admitted to the Brooklyn Cancer Institute over a 19-year period during which time 19,000 patients with all types of neoplastic diseases were admitted. This patient's lesion was also on the heel. Anderson[7] found only 10 cases of malignant melanoma in 14,000 surgical specimens at the John Gaston Hospital in Memphis. Five of the 10 cases reported were on the foot and 2 were on a finger. In 7 of the 10 cases the melanoma arose in skin which normally contained relatively little pigment, a fact which he believed had some bearing upon the problem. In only one of the 10 cases had there seemed to be a pre-existing benign lesion or mole (Fig. 1). In Anderson's review of these same 14,000 surgical specimens,

Fig. 1.—Foot of M. S., a 61-year-old Negress. The patient had always had a subungual nevus. It had become tender 3 months before this photo. It had been previously biopsied and proved to be melanoma. (This ultimate change in the nevus of course is expected, but a junctional nevus in the subungual position in the Negro is extremely rare.) This patient developed groin metastases 73 months after disarticulation of the third toe.

78 per cent of them coming from Negro patients, 12 malignant melanomas were found in white patients, giving an incidence in the white population 4 times that of the Negro race. He was of the opinion that the frequency with which malignant melanoma arose in the Negro on the foot from a traumatic incident was an important etiologic factor. Imler and Underwood,[33] however, asserted that melanoma was found on the soles in greater frequency in the Negro race, but that there was no basis to support the belief that melanoma is more apt to arise in areas of partial pigmentation. Enos and Holmes[24] reported 9 of 16 melanomas in the Negro on the lower extremity. Eight of them were on the feet. Morris and Horn,[40] reporting 113 cases in 1951 from the University of Pennsylvania, found the relative incidence of melanoma in the white population to be 4.4 times greater than in Negroes. In their series of 287 patients, 439 tumors were anatomically located. Of this number, 177 malignant melanomas

occurring in the Negro were primary on the feet (63 per cent). Of these 31 were on the sole, 8 were subungual, and 74 per cent (213) of the entire group were located on the lower extremity. They noted that the incidence in previously reported series in the world literature varied from 1.8 to 4.4 times as great in the white race as in the Negro. Of coincidental interest was the occurrence of 13 melanomas on the finger.

Location and Type of Lesion.—The location of the lesion on the feet shows a slightly higher group occurring on the right foot than on the left, with 65 lesions on the right and 56 on the left. More occurred on the dorsum of the foot, as indicated in Table II, than on the plantar surface, with 68 on the dorsum and 54 on the plantar surface.

TABLE II. MELANOMA OF THE FEET
Location of Lesion

LOCATION	LEFT	RIGHT	TOTAL	PER CENT
Dorsal surface				
Foot	5	9	14	11.5
Heel	9	8	17	13.9
Ankle	2	2	4	3.3
Toe	6	11	17	13.9
Toe, subungual	8	8	16	13.1
	30	38	68	55.7
Plantar surface		23	33	
Foot	10		14	27.0
Heel	7	7		11.5
Toe	3	4	7	5.7
	20	34	54	44.3
Total	50	72	122	100.0
Total number of cases during this 16-year period			917	
Total number of cases on the feet or ankle			122	
Distribution on the feet			13.3 per cent	

The presenting complaint of the patients varied greatly. The largest number, 19, described their complaint as a mole. Another 15 described a recent pigmented area. Fourteen patients complained of a recent ulcerating area; twice the lesion occurred after an injury. Seven of the patients complained of a blister or a "blood blister," and 13 patients simply noticed a lump or growth. Five patients complained of a pimple. Only 11 patients complained of a birthmark which had been changing in recent months (Table III).

Awareness of Pre-existing Lesion.—Inasmuch as junctional and compound nevi constitute the preponderance of nevi on the foot, it would be most interesting to correlate any information regarding subsequent development of melanoma from pre-existing nevi on the feet. Allen and Spits[4-6] stated that every melanocarcinoma of the skin or mucous membrane arises from a junctional or compound nevus, except, of course, the rare malignant blue nevus. However, in questioning patients there are a great many who have never noticed a pre-existing pigmented area or change, a fact which of course does not disprove the preceding statement. Ackerman[2] reported that 61.3 per cent of 75 patients noted origin of their melanomas in pre-existing nevi, Webster, Stevenson, and Stout[59] reported 65.4 per cent in 104 cases, Affleck[3] 83.8 per cent in

317 cases, Hall, Phillips, and White[26] 70.4 per cent of 159 cases, Howes and Birnkrandt[32] 54 per cent of 32 cases, although others have reported a lower incidence, such as Daland and Holmes[21] with 18 per cent of their cases. Becker[9] considers the incidence to be less than 25 per cent. Morris and Horn[40] noted a history of pre-existing mole in 2 of 9 Negro patients with melanoma.

TABLE III. MELANOMA OF THE FEET

PRESENTING COMPLAINT			
Mole	19	Pyogenic infection	1
Recent pigmented area	15	Painful red area	2
Ulcerated area	14	Injury to nail	2
Growth or lump	13	Nail fell off	1
Blister	7	Noticed node in groin	3
Birthmark	11	Burning sensation on heel	1
Pimple	5	Cracked area in skin	3
Hangnail	4	Injury due to burn	1
Wart	6	Injury due to cut	1
Pigmentation around nail bed	3	Callus	3
		No complaint	7

DURATION OF LESION*	NUMBER	PER CENT
Total	122	99.9
6 months and less	23	18.8
7-12 months	35	28.7
13-18 months	14	11.5
19-24 months	20	16.4
2-3 years	12	9.8
3-4 years	2	1.6
4-5 years	5	4.1
5-10 years	6	4.9
11-15 years	1	0.8
16-20 years	1	0.8
No time given	3	2.5

*The shortest time mentioned was 5 weeks.

Decker and Chamness[23] found a history of pre-existing nevi on the feet in 9 of 25 melanocarcinomas (36 per cent). Bickel, Meyerding, and Broders,[10] reporting 155 cases of melanoma of the extremity, found 40 cases with pre-existing history of a mole (32.3 per cent). In this series, only 30 of 121 melanomas on the feet (24.8 per cent) were apparently preceded by a mole of long-standing or a birthmark. Eighteen others related a recent history of a pigmented area or pigmentation around a nail. We assume that these tumors may have represented melanomas at the very onset. It seems difficult to reconcile the small percentage of people with melanomas arising on the feet who were never aware of preceding pigmented change in the area, especially since this site preponderates in incidence of junctional or compound nevi. Obviously, awareness of such pre-existing change and its existence are 2 entirely different matters. For instance, 3 people noted a node in the groin before noticing anything wrong with the foot. Undoubtedly there are many dermal nevi invisible within the narrow range of human vision, and such foci could well be the site of malignant change. As proof of this assertion, we have detected such occult moles by examining the skin in a dark room with a "cold" ultraviolet light. Per contra, completely excised early melanomas when minutely studied under the microscope do not always reveal vestiges of precursory benign nevi, under which circumstances one would not be able to deny categorically that some of these melanomas on the hands and feet could originate de novo.

The remainder of the patients complained of a wart on 6 occasions, 4 of hangnail, 3 of pigmentation around a nail, 2 of painful areas which proved to be melanoma, 2 of an ulcer developing at the site of a nail injury, one of a sore developing on a metal burn, and another of a nail that had fallen off. One patient complained only of a cracked area on the skin. Another similar complaint was that of a patient who had noticed a callus which proved to be a melanoma (Table III). The duration of the lesion varied greatly, and in several cases no change had been noticed during many years (Table III).

The diagnosis of each of these melanomas rested entirely with the pathologist on the basis of local excision, submitted slides after local excision, or excision of metastases. Slides of all cases reported cured of melanoma have been reviewed and confirmed by Drs. Sophie Spitz or Arthur C. Allen. It was interesting that one supposed long-term survival on review was found to have a blue nevus and the reputed metastases in the inguinal nodes proved to be phagocytized pigment.

Treatment of the Primary Melanoma.—All of the subungual melanomas or those on the digits were treated either by disarticulation at the metatarsophalangeal joint or amputation through a metatarsal bone. Most of the remaining tumors were treated by wide excision and the immediate application of a skin graft of intermediate thickness (Fig. 2). A 3 dimensional excision of equal extent is impossible in this area, but a minimal excision of several centimeters of normal skin around the primary tumor was usual. The subcutaneous tissue and deep fascia were included in the dissection except on the dorsum. If possible, the fascia overlying the extensor tendons was spared. Those patients whose melanomas were on the heel usually received a split thickness. Despite the usual feeling against this procedure, all had serviceable grafts if a small rim of the heel could be left to bear the greatest burden of pressure.

TABLE IV. MELANOMA OF THE FEET
Treatment Confined to the Feet

	NUMBER OF PATIENTS	AVERAGE TIME INITIAL SYMPTOMS TO LOCAL TREATMENT (MONTHS)	FROM LOCAL TREATMENT TO DEFINITIVE STATE (MONTHS)	TOTAL COURSE OF MELANOMA (MONTHS)
Alive and well	11	12.8	81.5	112.4
Dead, other causes	2	15	41.5	56.5
Indeterminate	9	20.2	38.4	50.6
No treatment after local excision elsewhere	1	29	34	70.3
Dead of melanoma	9			
Total	32			
Total in series	122—26.2 per cent local treatment only			
Absolute survivor rate	34.4 per cent			

Thirty-two of the 122 patients (26.2 per cent) had no further treatment than the control of the local melanoma. Eleven of these patients survived 5 years or more without further manifestation of melanomas, an absolute cure rate in this group of 34.4 per cent, which is quite comparable to Lund and Ihnen's figure of 38.9 per cent cure rate for melanoma treated by wide local excision alone. Table IV summarizes their course and tabulates the average time from

initial symptoms to the local operation, the average time from the operation to a definitive state, and the total course of the melanoma. Such data afford opportunity to study possible variations in the biology of the melanomas and serve as a comparison in the entire series.

The 5-year survivor rate of the entire group of 122 cases was 25.3 per cent (Table V) as opposed to 34.4 per cent for those patients whose care by fortuitous circumstances was confined to the feet. Since the surgeon has little

A.

B.

Fig. 2.—*A*, A malignant melanoma in a congenital nevus of the foot in a 34-year-old man. Six months before this photograph it began to grow and become lighter in color. *B*, Photograph after an adequate excision which included the deep fascia of the underlying area. This patient developed a solitary metastatic focus in a femoral node just over 5 years from the first operation. A radical groin dissection showed only one metastatic node.

control of the vascular spread of this cancer, his management of the lymph node bearing region is the second matter of important consideration. Of course, it is axiomatic that the primary melanoma is considered to be under control before considering treatment for the region nodal basin. In the period of this survey, the surgical philosophy assumed that the usual method of dissemination was by tumor emboli to the regional nodes. Inasmuch as early regional involvement was difficult to determine accurately by palpation, elective radical groin

dissections were performed. Whether or not the procedure was actually a therapeutic one or an elective one, usually from 2 to 10 weeks elapsed before the dissections were performed, although most were from 4 to 6 weeks after the local treatment was considered complete. Table VI presents, in part, an analysis of 56 cases of radical groin dissection in the control of 122 patients, as well as the various types of treatment used. In reviewing these cases, consideration was given to the apparent duration of the tumor before initial treatment, though it was frequently difficult to determine this period accurately because those melanomas arising in a pre-existing mole or birthmark presented such vague variations in appearances as to make it hard for the patient to assign a definite date of a specific change drawing his attention to the melanoma. Consideration is given from the time of apparent nodal involvement to the groin dissection and the postoperative course after the dissection. In thus assessing the life history of the melanoma, there might be a better index of the biologic activity of the tumor and better evaluation of therapeutic efficacy of surgery at various stages of the disease. This figure is often based upon the time a patient or his physician first observes nodal enlargement.

TABLE V. END RESULTS IN THE TREATMENT OF MELANOMA OF THE HANDS AND FEET (1935 through 1950)

	HANDS	FEET
Total number of patients examined	29	122
Indeterminate group		
Consultation only, no treatment	3	13
Dead within 5 years of other causes—No evidence of disease	1	2
Lost to follow-up under 5 years without recurrence of melanoma	5	12
Total	9	27
Determinate group		
Total number minus those of indeterminate group	20	95
Failures		
Dead as a result of melanoma	11	68
Alive with melanoma under 5 years	1	1
Alive without evidence of melanoma under 5 years since last treatment		2
Successful results		
Died free of melanoma over 5 years since treatment	1	1
Free of melanoma at 5 years or more, alive and well	7	23
Net 5-year results:		
Successful results determinate group =	40	25.3
Total number in determinate group	per cent	per cent

To Dec. 1, 1955.

In 31 patients considered by the examiners to present evidence of metastases in groin nodes at the time of admission to the hospital, metastases were substantiated 28 times and were not found on 3 occasions, a 9.7 per cent "false positive" error. On 10 occasions therapeutic dissections were performed because clinical signs became obvious during the course of follow-up examinations and all were proved positive. Therefore, in 3 of 41 patients there was a "false positive" diagnostic error of metastasis in groin nodes (9.7 per cent). This also may be taken to indicate a series of patients with obviously far-advanced cancer.

TABLE VI. MELANOMA OF THE FEET
Type of Treatment
(1935 through 1950)

Treatment confined to the local area	32	
Therapeutic node dissections	46	
Node dissections on admission		31
Node dissections during clinical course		10
Node dissections considered clinically negative but proved positive		5
Elective node dissection, no proved metastases	10	
Hip joint disarticulation with iliac and retroperitoneal node dissections	13	
Supracondylar amputations with radical groin dissection, nodes positive	3	
Amputations through thigh, leg, or foot	5	
No treatment or consultation only	13	
Total melanomas on the feet	122	

In the patients whose groin metastases became clinically obvious in the course of follow-up examinations, an average of 20.7 months elapsed before this was evident (Table VII), as compared to 15 months in an earlier series[48] and 14 months in Clark's experience.[19] This is a noteworthy period of time since frequent and close examinations of the groin are mandatory if elective dissections are not considered.

TABLE VII. THE ROLE OF THERAPEUTIC GROIN DISSECTION WITH NODAL METASTASES DEVELOPING DURING CLINICAL OBSERVATION
(1935 through 1950)

	NUMBER OF PATIENTS	AVERAGE TIME FROM APPEARANCE TO INITIAL TREATMENT (MONTHS)	FROM INITIAL TREATMENT TO GROIN DISSECTION (MONTHS)	FROM GROIN DISSECTION TO DETERMINATE STATE (MONTHS)	TOTAL COURSE OF MELANOMA (MONTHS)
Alive and well over 5 years	1	120	11	101	232
Alive and well under 5 years	1	6	63	7	76
Dead	8	22.7	16.6	20.7	48.9
Total cases	10	30	20.7		

In 5 patients the clinical signs favored freedom from metastases on initial examination when they were proved to be positive upon elective dissection, a "false negative" error of 8.2 per cent.

Therapeutic Groin Dissection.—There were 46 patients who underwent radical groin dissection for proved metastatic melanoma. Five (10.9 per cent) are alive and well an average of 126 months (or 10 years plus), 3 are indeterminate after a follow-up survey averaging 9 months, and 36 patients are dead with disseminated metastases occurring within an average of 18.4 months after the groin dissection and 38.8 months after initial appearance of their melanomas (Table VIII). The duration of postoperative survival of these patients eventually dying with the disease compares with the average figure given by Decker and Chamness[23] of 22 months in 11 patients having groin dissection after melanoma on the sole of the foot. However, it is not stated in this series whether or not all of these patients had regional node metastases proved. Lund and Ihnen[34] have found a 14.8 per cent 5-year cure for therapeutic regional dissections for melanoma.

TABLE VIII. THE ROLE OF THERAPEUTIC GROIN DISSECTION IN CONTROL OF MELANOMA OF THE FEET
(1935 through 1950)

	NUMBER OF PATIENTS	INITIAL SYMPTOMS TO FIRST TREATMENT (MONTHS)	LOCAL TREATMENT TO GROIN DISSECTION (MONTHS)	GROIN DISSECTION TO DETERMINATE STATE (MONTHS)	TOTAL COURSE OF MELANOMA (MONTHS)
Alive and well	5	34.0	4.0	97.2	126.8
Indeterminate	3	21.3	-	16.5	33.7
Alive without evidence of melanoma under 5 years since last treatment	2	18.5	38.5	37.5	43.5
Dead	36	17.5	6.6*	18.4	38.8
Total	46	—	37.7 per cent		
Total number of patients	122				
Absolute survivor rate	$\frac{5}{46}$	= 10.9 per cent			

*Estimated time for 27 patients.

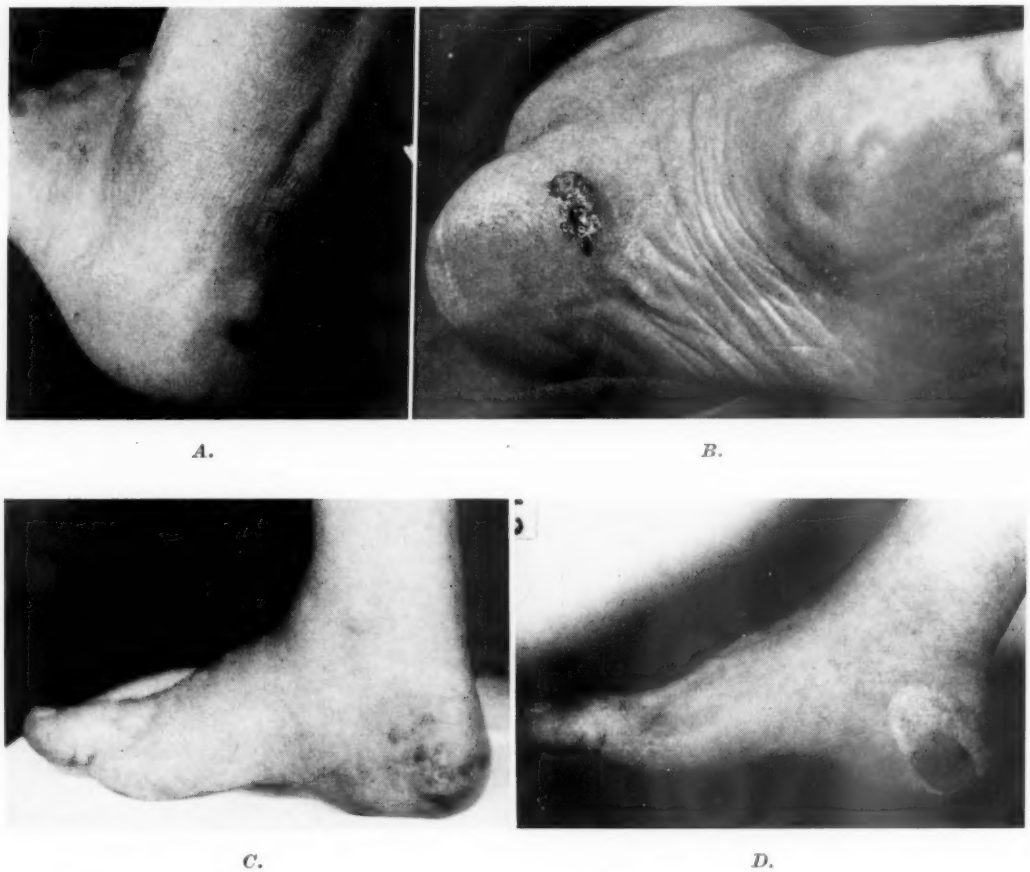
Groin Dissections Without Clinically Apparent Nodal Metastases.—Perhaps Brown and McDowell[15] have more precisely defined this issue of nodal dissections from an elective or prophylactic basis as "dissections for probable small metastases," but the time-honored acceptance of the former terms exonerates their continued use.

To regroup the patients who had radical groin dissections in order to consider the problem of those who had dissections without clinical signs of lymph node involvement, there are a total of 15 cases for consideration (Table IX, A). Five of 15 patients were 5-year survivors without evidence of melanoma (33 per cent of this group) as contrasted with the absolute 5-year survivor rate

TABLE IX. ELECTIVE GROIN DISSECTIONS IN CONTROL OF MELANOMA OF THE FEET
(1935 through 1950)

		NUMBER OF PATIENTS	AVERAGE TIME FROM INITIAL APPEARANCE TO INITIAL TREATMENT (MONTHS)	FROM GROIN DISSECTION TO DETERMINATE STATE (MONTHS)	TOTAL COURSE OF MELANOMA (MONTHS)
A. No Clinical Evidence of Metastases, Including Those Patients Both With and Without Proved Metastases of Melanoma					
Alive and well		5	19.8	138.5	156
Indeterminate		4	11	67	86
Dead		6	22.8	41.5	57.8
Total	15	Absolute survival rate		33 per cent	
B. Nodes Free of Proved Metastases					
Alive and well		4	21	155	176
Indeterminate		2	12	34.5	46.5
Dead		4	20	44.7	64.2
Total	10	Absolute survival rate		40 per cent	
C. Nodes Clinically Negative for Metastasis But Proved to Contain Metastatic Melanoma (Under 10 Weeks)					
Alive and well		1	5	71	76
Indeterminate		2	18	32.5	39.5
Dead		2	45.5	16	62.5
Total	5	Absolute Survival Rate		20 per cent	

of 25.3 per cent. By comparison, within this group no regional node metastases were discovered after radical groin dissection in 10 of these 15 patients (Table IX, B). Four of these present an average survival time of 155 months and are alive and well at the time of the study (Fig. 3, *A-D*). Two of the patients are indeterminate after an average of 34.5 months' follow-up, and 4 obtained an average survival of 44.7 months before dying of melanoma.



A. *B.*

C. *D.*

Fig. 3.—*A*, Heel of E. K., a 39-year-old woman with a 2-year history of a melanoma on the heel, measuring 10 by 13 mm. It was treated by wide excision and immediate application of a full-thickness graft. There were no palpable lymph nodes but an elective groin dissection was performed 5 weeks after primary operation. No metastases were found in the nodes. There was no evidence of recurrence at date of last examination over 11 years after first operation. *B*, Melanoma on foot of R. G., a 50-year-old woman. There has been little change in the appearance of this melanoma since it was first noticed 2 years previously. A wide excision followed by free full-thickness skin graft was followed 8 weeks later by elective groin dissection, without evidence of recurrence or metastases at time of last examination 15 years after primary operation. *C*, Foot of F. P., a 63-year-old man with an ulcerated nonpigmented melanoma of the heel and widely spaced pigmented satellite nodules. Elective groin dissection showed no evidence of nodal metastases. *D*, Foot of F. P., 12 years after excision and split thickness skin graft of what today would be considered inadequate proportions. Patient was free of apparent metastases when last operated upon at age 75.

In this series, 5 of the 46 patients with proved regional node metastases were not suspected clinically, an 8.2 per cent error as opposed to a 9.7 per cent "false positive" error in the same group (Table IX, C). Decker and Chamness found that in 10 operations considered as elective dissections, unsuspected metastases were discovered in 2 cases (20 per cent). Pack and Livingston,[50] in an

early series, found an incidence of 50 per cent of their cases with minute foci of metastatic melanomas which were clinically undetectable. Taylor and Nathanson,[57] in a more general study of all regional node basins, found that there was an incidence of lymph node metastases of 19 per cent of their cases in which the nodes were not palpable, but when the nodes were palpable they also proved to be innocent in 19 per cent. They add that the clinical failure to recognize involved nodes varies in the different node basins. They found a failure to recognize metastatic cancer in nodes in only 7 per cent with cervical metastases, 22 per cent with axillary metastases of melanoma, and 30 per cent with inguinal node involvement. In another general consideration of regional node dissection for melanoma, McCune[14] found that 7 of 12 (58 per cent) showed pathologic evidence of metastases though they were unsuspected clinically. This group of unsuspected groin metastases therefore represents a prime consideration in a yet unsolved problem of elective regional node dissections. In this small group of 5 cases, only one patient lived without evidence of melanoma over 5 years after treatment, and 2 died with the disease with the course on the average about the same as those who had dissections performed for obvious metastases.

Elective Groin Node Dissections for Melanoma.—McCune and Letterman[42] more recently have reported 36 cases of melanoma treated by radical groin dissections at the Walter Reed Hospital between 1942 and 1945. In these cases radical node dissections were performed in all of the cases regardless of the stage of disease as soon as possible after a pathologic diagnosis of melanoma had been determined. In order to determine the advisability of elective node dissections, the cases were divided into those in which the regional lymph nodes were enlarged at the time of the dissections and those that were not. These patients were followed for 10 years and, of 22 patients in the group with regional nodes enlarged at the time of the dissection, only 5 survived for 2 years and only one for 5 years. Of the 14 patients in whom regional nodes were not apparently involved, 7 showed evidence of metastases. Of this group 3 survived 8 years (43 per cent), and 5 (71 per cent) of the 7 without metastases survived over 10 years. This group, however, is not broken down as to the source of the primary melanoma, but on the basis of these findings these authors favor a routine regional node dissection before metastases are apparent, as do Meyer and Gumport[37] who have reported a group of 30 cases of groin dissection with an over-all survival of 46.7 per cent. Eighteen had positively involved nodes, with a 33.4 per cent survival, and 12 with groin nodes free of metastatic involvement had a 66.7 per cent survival. However, the 5-year survival figure of 20 of the 30 patients was reported as 25 per cent of 12 patients with metastases in nodes, and 62.5 per cent of those without metastatic melanoma in groin nodes.

We cannot understand these unusually favorable reports. In our attempt to assess the efficacy of radical groin dissections in this group of patients whose primary melanoma was only at one site, the 5-year survival for therapeutic node dissection for proved nodal metastases in the entire group has been shown to

be 10.9 per cent. The validity of this figure is corroborated by Catlin's[16] experience of 10 per cent 5-year control of metastatic melanoma in cervical node dissections for malignant melanoma primary on the head and neck region. His experience, like ours, would appear unfavorable in comparison with Meyer and Gumport's figure of 50 per cent 5-year survival with positive cervical nodes. Taylor and Nathanson,[57] however, only report an 18 per cent cure rate for melanoma metastatic to regional lymph nodes.

We have noted that in the 5 patients whose nodes were clinically free on admission but whose nodes were proved to have clinically undetected metastases, one (20 per cent) survived for 5 years and was well. Of the 10 patients who had node dissections without metastases being found (6 without apparent and 4 with presumed but unproved clinical evidence of metastases), the 5-year control figure was 40 per cent (Table IX, B). In grouping these 2 together (Table IX, A) 5 of 15 patients survived without evidence of spread of the melanoma (33 per cent). It is especially to be noted that in this group the life span of those patients dying with melanoma is 23 months longer after groin dissection than for those whose metastases were discovered by dissection before they were clinically apparent, suggesting a quantitative phenomenon in the process of dissemination of the melanomas. Lund and Ihnen[34] have expressed a similar viewpoint in their excellent analysis of this problem in stating that "the degree of metastatic involvement of excised lymph nodes has been considered important in the prognosis of other tumors, such as carcinomas of the breast or colon; it probably has similar importance in malignant melanoma, especially if there is minimal or advanced metastatic involvement." They noted that this information is rarely given. An analysis of nodal metastases in the groin comparable to Adair's[1] analysis of axillary metastases found after radical mastectomy is not available.

This series, therefore, would indicate that those patients with metastatic melanoma in the groin, whether clinically apparent or not, have an ominous prognosis, but the extension of life apparent in those who die after a dissection for nodes harboring melanoma not clinically apparent would indicate that elective node dissections may be of avail in extending life in most and may occasionally offer a cure. It appears to us that it is evidence in support of the procedure described by Handley[28] as "an intelligent anticipation of the trend (which) may sometimes avail to stay the finger of fate." A more recent and indeed most critical analysis of the problem has been epitomized by Lund and Ihnen in stating that "delay in dissection was not very successful and there is suggestive evidence that earlier lymph node dissections might have been beneficial."

In any consideration of the treatment of metastatic melanoma, the views of Handley[28] are germane since the subject of his original investigation had melanoma over the insertion of the tendo achillis on the heel and presented no recurrence after an excision. He endeavored to show that centrifugal permeation of the lymph vessels around a focus of melanoma, whether primary or secondary, is the primary factor in dissemination and that embolic spread by

way of the blood stream is a later event, dependent upon the infiltration and invasion of the veins and arteries from concomitantly permeated lymphatics. While the theoretic concept of treatment pursuant on this assumption is not to be controverted, in the hand and on the foot its practicality is open to conjecture as far as our figures are concerned at this time as is shown in the following discussion.

A. *B.*

Fig. 4.—*A*, Heel of R. M., a 57-year-old woman with a 6-month history of a small black lump on the left heel. Two months after appearance it began to pain. Two weeks before this photograph the lesion began to grow rapidly and ulcerate. It measured 4 cm. in diameter. An amputation below the knee was performed, and 6 weeks later a therapeutic groin dissection was performed. *B*, Adductor side of thigh and the amputated leg with the manifestation of diffuse lymphatic permeation by melanomatous metastases (satellitosis). This photograph was taken 17 months after groin dissection for numerous nodal metastases. A hip joint disarticulation was done at this time and the pathologist reported diffuse lymphatic permeation of the melanoma. The patient died of generalized metastases 18 months later.

Local Recurrence and Diffuse Metastases in the Extremity.—It was during the course of the management of these patients that the event of diffuse dissemination in the extremity, especially along the saphenous vein and its accompanying lymphatic tributaries, either with or without local recurrence, often became evident as a complication of a regional node dissection performed without dissection in continuity of the intervening lymphatic channels. After the primary plan of surgical management had been carried out on these melanomas, whether for local recurrence or for primary lesions and whether the initial management included regional node dissection or not, there were 20 cases (16.5

per cent) showing either local recurrence and/or diffuse metastases in the extremity (Fig. 4). Fourteen times there were diffuse recurrences in the extremity or in the groin scar after regional node dissection (14 of 56 dissections, 25 per cent). Only one of these failed to show metastases of the melanoma in the groin at the time of radical iliac node dissection. All of these patients died.

There were 6 local recurrences of melanomas after control of the primary lesion, 5 after groin dissections for proved metastases and one after no pathologic proof of groin metastases was established. The latter case had a 5-year survival after a mid-thigh amputation for recurrent melanoma although this patient had proved popliteal node metastases as well as vein invasion (Fig. 5, *A*). Four melanomas recurred in the groin or abdominal wall after regional node dissection and in 3 of these instances there were proved nodal metastases. All of these patients died with dissemination of the disease.

Fig. 5.—Locally recurrent melanoma of heel of B. F. who survives over 5 years without recurrence or metastases after mid-thigh amputation and groin dissection. There were popliteal node metastases and vein invasion at time of operation, which indicated a very ominous prognosis.

Melanoma on the Feet With Simultaneous Nodal Metastases in the Groin.—Melanoma of the foot with simultaneous nodal metastases in the groin is generally believed to give a universally ominous prognosis. Theoretically it would seem that the principle of an "in continuity" dissection would demand a retroperitoneal dissection with disarticulation, or better yet, hemipelvectomy. Table X does not present strong evidence in support of this concept and does not confirm Bowers'[14] consideration of quarterectomy in its application to malignant melanoma in which he has stated that in melanomas of the dorsum of the foot, leg, or thigh, ordinary thigh amputation with or without dissection of lymph nodes is to be condemned. This clinical setting occurred 25 times (20.5 per cent) in this experience with 122 patients. This includes those patients with either clinically obvious and proved or clinically undetected but proved metastases. Five times the melanoma was a recurrent one on the feet. Analysis of Table X indicates that an absolute 5-year control of 12 per cent does not present a hopeless condition. One patient worthy of note had a groin dissection for metastases

in the groin 62 months before the primary tumor was discovered beneath the toenail and yet has not had intervening lymphatic metastases (Fig. 6). There is no doubt that combined local treatment and groin dissection in these patients may invite local recurrence in at least 25 per cent of these patients, but the possibility of recurrence in the scar of quarterectomy in this clinical setting is also as great. Data of Table X would indicate the life history of these patients with melanoma is quite similar to the over-all picture of therapeutic groin dissection without simultaneous consideration of the primary site. For the sake of comparison of this problem either with the first clinical problem presenting as a yet uncontrolled primary with regional node metastases or as a local recurrence with groin metastases, analysis of the life span of those dying with melanoma would not indicate a great difference in the figures *if* the 5 patients with local recurrence are excepted. There were *no* cures in this latter group, however.

Fig. 6.—Toe after excising residual part of nail to excise a small "granulating" point on toe which was considered suspicious of nonpigmented melanoma which it proved to be. This 50-year-old patient, however, had had a groin dissection for what was felt to be a spindle-cell sarcoma 78 months before. One year before this a doctor had partially removed the nail for an "eponychia" which healed and did not recur. This was probably a small melanoma that did not recur until 88 months later. Though recurrence has followed amputation of the toe, diffuse recurrence in the extremity has not followed.

TABLE X. MELANOMA OF THE FEET WITH SIMULTANEOUS INGUINAL NODE METASTASES (1935 through 1950)

	NUMBER OF PATIENTS	AVERAGE TIME FROM INITIAL SYMPTOMS TO COMBINED THERAPY (MONTHS)	FROM LOCAL TREATMENT AND GROIN DISSECTION TO DETERMINATE STATE (MONTHS)	TOTAL COURSE OF MELANOMA (MONTHS)
Alive and well over 5 years or dead without melanoma	3	16	89.6	109
No treatment	3	6.6	16	22.6
Indeterminate after treatment	1			
Alive with disease over 5 years	1	16	62	78
Dead from melanoma	17	15.23	21.05	36.08
Total cases / Entire cases	25/122 = 20.5 per cent of patients with simultaneous manifestations			
5-year cure rate = 12 per cent				

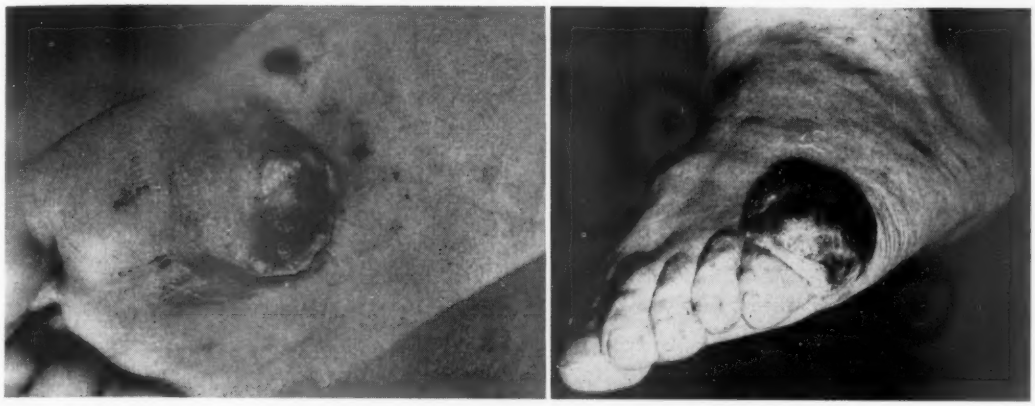
Major Amputations for Melanoma.—Because of the impression of a high incidence of recurrences in these patients and because of diminishing hope of control of the melanoma by radical groin dissection after regional node metastases had been proved, the opinion was entertained that experience obtained during the first part of this study would indicate that a program of major amputations for those melanomas with proved inguinal node metastases might be in order. In order to attempt a greater salvage rate in these people with an extremely ominous prognosis with proved inguinal node metastases, the plan for operative management then became that of a wide exposure of the retroperitoneal region to see the extent of lymph node involvement high in the iliac chain. If the nodes at the highest level of the dissection proved negative for metastatic melanoma on frozen section and it was at least grossly possible that all evident melanoma could be removed, a high iliac dissection was performed, stripping the lymphatics along the iliac vessels, ligating the external iliac artery and vein, and finishing the procedure with a disarticulation at the hip joint, preserving a long posterior flap and thereby sacrificing all the tissues in the groin. Closure was effected from the posterior aspect of the wound. This is a modified Boyd[12] type of disarticulation since it enters the area of the superficial and deep femoral node compartments of the thigh to the least extent and the greatest part of the operation is performed by direct approach to the femoral structures at the level of the groin. In some instances the dissection has been obviated by the use of hemipelvectomy.

TABLE XI. THE ROLE OF THE HIP JOINT DISARTRICULATION AND ILIAC NODE DISSECTION IN THE CONTROL OF MELANOMA OF THE FEET
(1935 through 1950)

	NUMBER OF PATIENTS	APPARENT ONSET OF MELANOMA TO LOCAL TREATMENT (AVERAGE IN MONTHS)	LOCAL TREATMENT TO HIP JOINT DISARTICULATION (AVERAGE IN MONTHS)	HIP JOINT DISARTICULATION TO DEFINITIVE STATE (AVERAGE IN MONTHS)	TOTAL COURSE OF MELANOMA (AVERAGE IN MONTHS)
Alive and well	1	5	1	62	68
Dead with melanoma over 5 years	1	36	36	65	105
Dead with melanoma under 5 years	11*	22.2*	17.7*	17.9	47
Total number of patients	13				

*Average of 9 patients.
Five patients treated by hip joint disarticulation on admission.
Group includes only one patient with no metastases found in nodes. Five-year survival without evidence during interval 18.1 per cent (2 of 11).

Table XI summarizes the beginning of this operative experience in the use of amputation or disarticulation in the control of melanoma. In 12 patients with proved groin metastases, one patient survived over 5 years after groin dissection and disarticulation though he died shortly after this from a cerebral vascular accident which seemed compatible with a cerebral metastases of the melanoma, although an autopsy was not obtained. The cause of death has



A. *B.*

C. *D.*

Fig. 7.—*A*, The foot of a 60-year-old man who had been watching this lesion expand from a "wart" in the preceding 5 years. A supracondylar amputation was done and followed by a groin dissection one month later for metastatic melanoma. Three years later a cancer of the sigmoid colon was resected. The patient died of pulmonary metastases 129 months after groin dissection but autopsy was not performed and which cancer caused death could not be determined. *B*, Foot of a 59-year-old man, S. G., who had a 2-year history of a "sore" on the fifth metatarsal which had been excised 6 months before and which rapidly regrew. A tarsometatarsal disarticulation was performed and shortly after a biopsy of a suspicious node in the groin showed no metastases. The patient died of diffuse metastases 16 months later. *C*, Sole of foot of E. J., a 70-year-old Negro woman, who had always had a mole about the size of a quarter (25¢) on this area. It was excised 3 months before this photo, and it recurred within one month. Because of recurrence and because no regional nodes were palpably suspicious of harboring metastases, mid-thigh amputation was performed after preliminary ligation of the superficial femoral vein. There were subcutaneous epidermal metastases at a radius of 2.5 cm. from the border of the tumor. No further evidence of melanoma was found at last examination 6 years after amputation for recurrence. *D*, Foot of A. McL., a 53-year-old woman with a locally recurrent melanoma. It had been present for 8 years before it was excised and the patient was given 8 x-ray treatments. Recurrence is of 3 months' duration and would appear to be a case with a poor prognosis. Amputation was through the middle third of the leg. The patient is alive and apparently in good health but classed as indeterminate about 5 years after operation. (Private patient of Dr. Gordon McNeer.)

been considered to be metastases. One patient survived without melanoma evident when explored for ovarian cancer 62 months after hip joint disarticulation. In all of these melanomas the initial lesion on the foot had recurred before metastasizing. In at least 3 there were metastases in the intervening lymphatics between the primary site and the groin, and another had one nodular metastasis in the lower leg. All of these cases represent most unfavorable settings to attempt control of the melanoma. One other patient had metastases in the skin of the leg but proved to have no nodal metastases but is included in this series as at least having lymphatic metastases. This patient survived the operation 36 months. These 13 cases presented no favorable surgical salvage rate of a very ominous clinical problem and further experience of this procedure awaits evaluation in a subsequent study. Whether or not earlier operations of this magnitude in melanoma would prove it to be no more malignant than cancer of the breast or uterus, as envisioned by Handley,[29] remains to be determined, but at this time such a statement is an unproved assertion. Mention should be made of Bickel, Meyerding, and Broders[10] who have considered the role of amputation in melanoma of the extremities. In their survey of 91 traced cases, in which amputation had been the mode of therapy in 22 instances, there was a 21.1 per cent 5-year survival. This group is not discussed in relationship to the type of amputation or the site of melanoma, but in the same series a 29.8 per cent survival rate of 63 cases treated by excision alone would indicate no great increase in the salvage rate. De Cholnoky[22] has stated that melanoma of the fingers, toes, and heels can be effectively eradicated by amputation followed by regional node dissection. Handley[28] epitomized the role of early amputation by stating that "When malignant melanoma arises in the digits, amputation should be performed at once . . . . when a melanotic growth occurs on the hands or feet above the digits, the question of amputation will always arise, but in early cases excision of the growth, even though it be attended by greater risk of recurrence, will usually be the operation of choice."

In 3 instances, supracondylar amputation with regional node dissection in continuity did not control the melanoma in any instance. One patient survived 139 months before dying of pulmonary metastases, possibly from the melanoma, although the patient had had a resection and a primary anastomosis for cancer of the sigmoid colon in the meantime (Fig. 7, *A*).

Four times amputations were performed in the site of election either in the thigh or leg for recurrent melanomas of the foot, and one had a Lisfranc amputation (Fig. 7, *B*). Two were alive and well over 5 years, one despite vein invasion being demonstrated by the pathologist (Figs. 5, *A* and 7, *C*), and one indeterminate at 5 years (Fig. 7, *D*).

Factors Influencing Prognosis of Melanomas of the Feet.—In consideration of the factors influencing prognosis, the relationship of the size of the melanoma was studied in 81 patients in which size had been determined or at least estimated. Seventeen patients had melanomas 1 cm. or less in diameter. Five of these (29.4 per cent) represent the absolute survivorship. Six of 29 patients had tumors of 1 to 2 cm. (20.7 per cent absolute survivorship), 2 of 9 with

tumors of 2 to 3 cm. (22.2 per cent), and 2 of 11 with melanomas of 3 to 4 cm. in size (18.1 per cent). One patient of 10 (10 per cent) with a tumor of 4 to 5 cm. in diameter survived without recurrence of melanoma over 5 years, whereas not one patient of the 5 with melanomas over 5 cm. in diameter survived. There is apparently no great variability in prognosis for melanomas under 4 cm. in diameter except for those of 1 cm. or less which have the best prognosis (29.4 per cent). There were 4 instances in which the pathologist noted vein invasion. Three of these patients died of melanoma, but one survived over 5 years without evident dissemination. The average delay in treatment of those surviving and those dying was about the same and did not appear to be a contrasting factor in prognosis. Ulceration, or its absence, was not specifically noted often enough to consider its influence on prognosis, although it has been shown to be a factor in other considerations.[5, 58]

End Results in the Treatment of Melanoma of the Feet.—There were 32 patients whose treatment was confined to the foot; of these 11 (34.4 per cent) survived over 5 years without recurrence or metastases. Three had subungual melanomas and were treated by digital amputation, whereas the remainder had wide excisions with skin grafting. Two patients died of other causes in less than 5 years and without evidence of melanoma. One patient was followed expectantly after treatment elsewhere and died from metastases. Of the 9 patients who died from melanoma, the average period of growth of the melanoma until treatment was instituted was 29 months, and an average of 34 months elapsed from treatment to death. The average course of the apparent life history of these melanomas was 70.3 months.

It has been apparent that if expectant observation of a melanoma locally controlled was elected as a course of treatment, an average period of 20.7 months elapsed before metastases became apparent in 10 patients of the group. Only one (10 per cent) of this group survived over 5 years without recurrence or dissemination of the melanoma after groin dissection became an obvious necessity.

Forty-six (37.7 per cent) of the group had therapeutic groin dissections, and only 5 (10.9 per cent) achieved successful results from their treatment. Of 36 patients dying with uncontrolled melanoma, the life history of the tumors spanned 38.8 months, indicating a tumor about twice as virulent (on the duration of its life course) as those tumors of patients dying with melanomas presenting primarily as a problem of local treatment.

When an anticipatory dissection of the groin was done without clinical evidence of metastases, even though the pathologist found undetected metastases, the absolute survival rate for a small group of 5 patients was only 20 per cent. Two patients of this group who died had melanoma running an apparent total course of 62.5 months. For comparative purposes, in review of those patients who died after either local treatment or local treatment plus groin dissection for what was pathologically verified as locally confined melanoma only, it is found that of 9 patients dying after having local treatment only the total average span of melanoma was 70.3 months as compared to an average of 64.2 months for 4 patients who died after an elective node dissection

proved freedom of undetected nodal metastases. Yet in each one of these groups the absolute survival rate was from 2 to 4 times better than the fate of those who had groin dissection for clinically apparent and proved metastases. These data at least indicate that nothing is to be lost in anticipating the nodal spread that will be a therapeutic problem in at least 50 per cent of those patients with melanoma arising on the feet. This corroborates Sugarbaker's[56] statement that "There can be little argument that cancer in lymph nodes is best treated while still in its subclinical stage."

One rather startling finding has been that if treatment must be given to a primary or a recurrent melanoma on the foot with obvious groin metastases and without intervening lymphatic metastases, a problem apparent in 20.5 per cent of the patients (Table X), a 5-year survival rate of 12 per cent for such groin dissections still occurs in the condition which has been felt to be the absolute reason for hip joint disarticulation with a retroperitoneal dissection. Yet this treatment does give a 25 per cent chance of diffuse recurrence in the extremity, the hazard of maintaining ambulatory capacity. However, disarticulation with a retroperitoneal node dissection does not improve the prognosis in this condition, since this group has had only an 8.3 per cent successful cure rate.

Consideration of these data indicates but does not prove a better survival rate for anticipated surgical control of probable but unapparent metastases in the groin and also indicates even a longer survival period for those eventually succumbing to their melanoma. We cannot confirm some of the more recent energetic and salutary results of elective nodal dissection and believe that our detailed experience confirms a more traditional and universal experience under these conditions.

Recapitulation of Experimental Therapy.—In the treatment of a tumor which at this solitary site may stay confined locally (as far as surgical expectancy is concerned) in about 26 per cent of the patients, perhaps more attention should be given to the fact that 62 patients (50.9 per cent) eventually had proved regional node metastases at a time when surgical dissection with or without other modes of treatment was in order. Even years ago in a series of elective groin dissections, a disconcerting number of occult metastases were found in femoral and inguinal nodes which were not clinically suspicious. We have not solved the problem pertaining to the sequence of management of the lymph nodes in the groin, that is, before or after metastases become apparent from melanomas primary on the feet. One certainty is that the groin dissection should never precede the surgical treatment of the primary pedal melanoma.

In an attempt to answer this question to the satisfaction of ourselves and the betterment of our patients we have been employing various clinical trials, namely:

A. During the 12-year period of 1935 to 1947, melanomas of the feet with known metastases to the groin were usually treated by excision and skin grafting or digital amputation for the primary lesion and radical groin dissection with preservation of the leg. The 5-year definitive cure rate for melanomas so

treated was never better than 14 per cent. An adequate conjoined superficial and deep groin dissection inevitably produces some degree of blockage and lymph stasis, which is the worse possible setting for residual melanoma in the leg, resulting in diffuse melanomatosis by lymphatic tumor permeation. Amputation after this accident has proved to be futile.

B. In a series of patients without nodal enlargement, the primary melanoma on the foot was treated by radical local surgery. Six weeks later a superficial groin dissection was performed for individual analysis of the nodes. The time interval was arbitrarily selected with the theory (not based upon knowledge) that melanoma emboli in transit would have an opportunity to reach the nodes and be enmeshed in the nodal filter.

C. In the most recent series, the melanoma on the foot has been treated by wide excision with skin grafting or partial or digital amputation with an immediate superficial groin dissection at the same operative seance and on an elective basis. A frozen section analysis of the groin nodes is requested, with acceptance of the limitations imposed upon the pathologist. If metastases are found, a radical groin dissection is completed, but, if not, the superficial groin dissection is deemed adequate. This treatment seems a preferable mode of therapy since over half of these patients with melanoma of the feet are going to come to a groin dissection during their clinical course. This anticipates the interval of expectant observation of the regional nodes, and, for those who eventually develop metastases, an average of 20 months of living with melanoma before it is clinically detected is saved. The treatment is supported by the best salvage rate of the proposed systems of treatment.

D. The lower extremity was amputated with retroperitoneal lymph node dissection for melanoma of the foot with proved metastases to the groin. Sacrifice of the limb has never been done until metastases were demonstrated in the femoral or inguinal nodes. The usual procedure was to perform a retroperitoneal iliac and obturator node dissection with hip joint disarticulation. Hemipelvectomy was performed when the suprainguinal nodes were fixed within the pelvis and therefore not dissectable. Cure has been obtained once to date, which is not necessarily a criticism of the procedure but rather an admission of the fatal tardiness of its application. In the greater number of cases wherein hip joint disarticulation was performed, the operation was one of desperation rather than of hope, because some patients had recurrent melanomas on the feet and others had melanotic metastases in the tissues intervening between the foot and groin. With these settings, the efficacy of retroperitoneal dissection with hip joint disarticulation remains to be proved, yet it would appear that this must be done in an earlier phase of the disease than in the past if the end results of its application are to be accepted as an improvement.

From these clinical experiments, the results of which have been given statistically in foregoing tables, one fact becomes evident. We are still uncertain about the principles of conjoined management of melanomas of the feet metastatic to the groin. Is it proper to preserve clinically uninvolved lymph nodes in the groin as somewhat efficient catchments for lymph-borne metastases from melanoma of the foot as long as the therapist is uncertain of the cure of the

primary pedal melanoma following his surgical treatment? Is it asking too much for any cancer as malignant as the melanoma to spread all the way from the foot to the groin through the myriad of lymphatics with their sluggish circulation and for none of the cells to become deposited in the numerous lymph nodes intercalated along these pathways? Rouvière[51] has shown that the lymphatics of the heel and lateral aspect of the sole penetrate the deep fascia and course upward along the deep vessels emptying into the popliteal nodes and others between the foot and the groin. Furthermore in the dissection of amputated lower limbs the deep and hidden melanotic metastases are often found along these inner pathways.

MELANOMAS OF THE HANDS

Of the 917 patients with melanoma of various parts of the body who were examined and treated at the Memorial Cancer Center during the 16-year period from 1935 through 1950, there were only 29 patients with melanoma occurring on the hand (3.2 per cent). It is apparent that it is difficult to accumulate enough cases in this location to have statistical significance. Stout[55] has previously presented a series of 22 melanomas in this region before the American Society for Surgery of the Hands. In our series of 29 cases, the slides were no longer available for review in 8 of the cases, although they were originally

TABLE XII. MELANOMA OF THE HANDS
Age, Sex, and Racial Distribution

AGE IN YEARS	TOTAL	MALE	FEMALE
Total	29	12	17
11-20	1	1	0
21-30	2	0	2
31-40	6	2	4
41-50	5	1	4
51-60	10	4	6
61-70	4	4	0
71-80	1	0	1
Range: 17 years (Male) to 74 years (Female)			

SEX	NUMBER	PER CENT	RACE	NUMBER	PER CENT
Male	12	41.4	White	26	89.7
Female	17	58.6	Negro	1	3.4
			Not stated	2	6.9

TABLE XIII. MELANOMA OF THE HANDS
Location and Duration of Lesion

LOCATION	NUMBER	PER CENT	DURATION	NUMBER	PER CENT
Total	29	100.0	Total	29	99.8
Dorsal surface	23	79.3	6 months or less	1	3.4
Hand	6	20.7	7-12 months	6	20.7
Wrist	1	3.4	12-18 months	1	3.4
Fingers	3	10.3	19-24 months	6	20.7
Subungual	13	44.8	2-3 years	3	10.3
Palmer surface	6	20.7	3-4 years	3	10.3
			5-10 years	6	20.7
			16-20 years	1	3.4
			Not recorded	2	6.9

Total number of cases during this 16-year period 917.
Total number of cases on the hand or wrist 29.
Percentage distribution on hands 3.2 per cent.

verified in the Department of Pathology and all long-term survivors were able to be restudied. Two other cases were excluded for lack of adequate histologic evidence.

Twenty-six (89.7 per cent) of the 29 patients were white; only one was Negro. In 2 cases no race was recorded. The frequency of age distribution was greatest in the sixth decade of life (10 cases). The age and sex distribution revealed 17 women (58.6 per cent) and 12 men (Table XII). The dorsal surface of the hands or fingers predominated, with 79.3 per cent of the melanomas occurring on this surface (Table XIII). The preferable treatment consisted of wide excision and skin graft for melanomas of the hand or wrist and amputation of the digital melanomas.

A. *B.*

Fig. 8.—*A*, Hand of J. Z., a 60-year-old man, with the history of a blister appearing 18 months before which turned blue and persisted. He removed it with a razor blade and it healed. It recurred 6 months before this photograph. *B*, Photograph after an index-ray amputation for melanoma, covering the defect with tissue from the dorsum of the hand. No further trouble had been found in 6 years of follow-up care.

Local Treatment of Primary Melanoma of the Hand.—Three of the 29 patients had no treatment; 2 of these refused major amputations, and one failed to return after biopsy of an ulcerating subungual lesion. Ten patients had treatment confined to the hand only. Five had amputations for subungual melanomas, and a sixth had an amputation through the base of the second metacarpal for a melanoma over the palmar aspect of the head of the metacarpal (Fig. 8). Two of these patients survived without evidence of melanoma over 5 years. Three had excisions, but 2 died soon of melanoma. One patient with a very limited local excision of a melanoma had no further evidence of melanoma in 9 years of follow-up examinations. Four of the 10 patients who had treatment confined to the hands were 5-year determinate cures (40 per cent).

Axillary Metastases From Melanoma of the Hand.—Fourteen of the 29 patients had axillary dissections. Of 8 patients who had elective dissections performed under 4 weeks after treatment of the primary melanoma on the hands, only one proved to have clinically undetected melanomatous metastases (12.5 per cent), although there were 2 other patients whose nodes showed only phagocytized melanin present without any tumor cells. One of these died 8 months later of cerebral metastases, while the other is alive and well over 84 months later. The first patient died of metastases 5 years after axillary dissection. Of the remaining 5 cases of elective axillary dissection without metastases being found in the nodes, one is alive and well 16 years later (Fig. 9) and another 5½ years later. One patient is indeterminate at 11 months. The 3 remaining in this group died of melanoma 8, 21, and 55 months, respectively, after the axillary dissection. The incidence of undetected metastases in this group of elective dissections is not as high as that mentioned in a previous report of Pack, Scharnagel, and Morfit[48] in which 5 (50 per cent) showed microscopic evidence of metastases though none were apparent clinically.

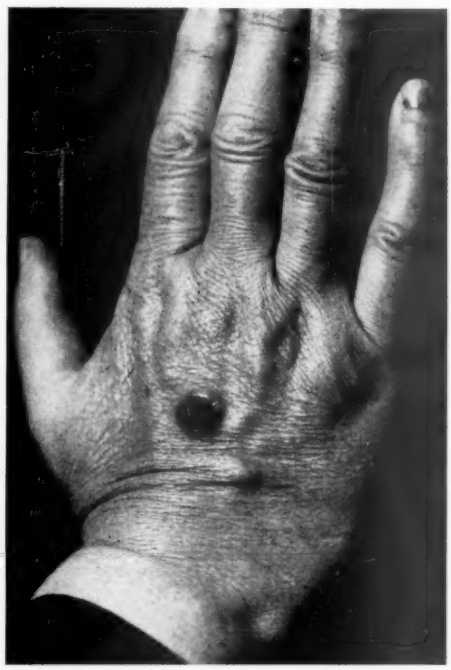


Fig. 9.—Hand of D. B., a 53-year-old woman, with a lifelong history of a black flat lesion on the dorsum of the hand, but which had been increasing in size since injuring it one year previously. It was a melanoma and was treated by excision and skin graft and subsequently elective axillary dissection. Patient is a 16-year cure.

There were 6 node dissections performed for what was apparently metastatic melanoma, and only one failed to show metastases in the nodes (Fig. 10). One patient died from a separate primary lung cancer 122 months after a local excision of a recurrent malignant blue nevus on the dorsum of the hand with nodal

TABLE XIV. THE ROLE OF ELECTIVE NODE DISSECTION IN THE CONTROL OF MELANOMA OF THE HANDS

	NUMBER OF PATIENTS	INITIAL SYMPTOMS TO FIRST TREATMENT (MONTHS)	LOCAL TREATMENT TO AXILLARY DISSECTION (MONTHS)	AXILLARY DISSECTION TO DETERMINATE STATE (MONTHS)	TOTAL COURSE OF MELANOMA (MONTHS)
Alive and well	3	11.3	Simultaneous	114	190
Indeterminate	1	48	1	11	65
Dead	4	26	Simultaneous	36	82.7
Total	8				
Absolute 5-year survival rate—37.5 per cent.					
THE ROLE OF THERAPEUTIC DISSECTIONS					
Alive and well melanoma over 5 years	1	72	1	97	170
Alive, presumably with melanoma	1	12	88	122	218
Dead	4	58.5	2.2	40.5	99
Total	6				
Absolute 5-year survival rate—16.6 per cent.					

A. *B.*

Fig. 10.—*A*, A nonpigmented subungual melanoma of the left middle finger of a 68-year-old man. He had noticed a "cracked-nail" 3 months before. The finger was amputated through the metacarpal and an axillary dissection was performed for a rather large node, clinically considered metastatic melanoma, but no metastases were proved. *B*, Satellitosis in arm taken 4 years after operation. The patient also had pulmonary metastases at this time.

metastases in the axilla (Fig. 11, *A*). One was alive after excision of a recurrence in the arm and a separate primary melanoma 9 years after axillary dissection (Fig. 12). The 4 patients who died after axillary dissection averaged 40.5 months survival (Table XIV).

Malignant Blue Nevus of the Hand.—This second patient is worthy of further comment since a malignant blue nevus is extremely rare. Montgomery[38] in 1952 reported his first experience in which a blue nevus underwent malignant

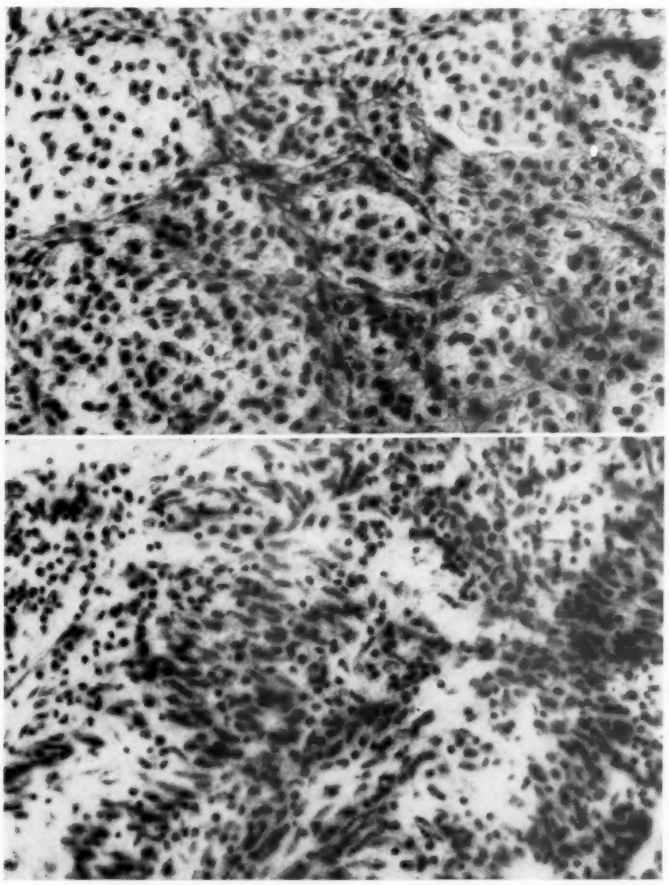


Fig. 11.—*A*, J. McD. Photomicrograph of a malignant blue nevus recurring on the dorsum of the hand showing spindle cells. There was no involvement of the epidermis itself. *B*, J. McD. Photomicrograph of an axillary lymph node metastasis showing spindle-shaped cells and collection of melanin pigment.

change. This patient had a blue nevus on the foot with metastases in the groin and was alive 3 years after the metastases occurred. He mentions one or 2 other cases and has more recently had another such case with evidence of metastases.[39] Furthermore, Gilston[25] has noted a patient whose primary lesion contained elements compatible with the diagnosis of Jadassohn-Tieche nevus. The cellular blue nevi and also malignant blue nevi have been discussed by Allen and Spitz,[5] who reported 4 instances of malignant blue nevi from the Memorial Cancer Center. One of these patients treated by us was a 52-year-old man who presented a locally recurrent malignant blue nevus of the hand and axillary

nodal metastasis. He had an excision of the tumor on the dorsum of the hand and axillary dissection. Fig. 11, *A* is a photomicrograph of the recurrent tumor which shows irregular fascicles of spindle cells without any involvement of the epidermis. There were axillary lymph node metastases present at the time of the recurrence (Fig. 11, *B*). The patient survived almost 15 years and died of bronchogenic cancer without evidence of melanoma.

A. *B.*

C.

Fig. 12.—*A*, Malignant melanoma of the index finger of a woman of Indian-Negro descent, age 59. This melanoma had been present for one year. *B*, A recurrence in the skin of the epitrochlear area 16 years after potential cautery amputation. *C*, Photograph taken one year after previous one showing both a local recurrence as well as a separate primary melanoma showing junctional changes in the area around the new primary.

Long-Term Survivors With Persisting Melanoma of the Hand.—The remaining 2 patients who had therapeutic axillary dissections are doubly worthy of note because of the long-term survival (20 and 15 years respectively) with metastatic melanoma and also the appearance in both patients of second primary melanomas. These patients also show that although melanoma is a highly malignant tumor occasionally it presents long periods of apparent symbiosis with the patient and manifests the fact that 5-year cures are not synonymous with absolute cures. Allen and Spitz,[5] from this same source material, have noted that 13 per cent of the patients with melanoma died of metastases 5 years or longer after the initial treatment of the tumors, 11 per cent survive 5 to 10 years, and 2 per cent 11 to 24 years.

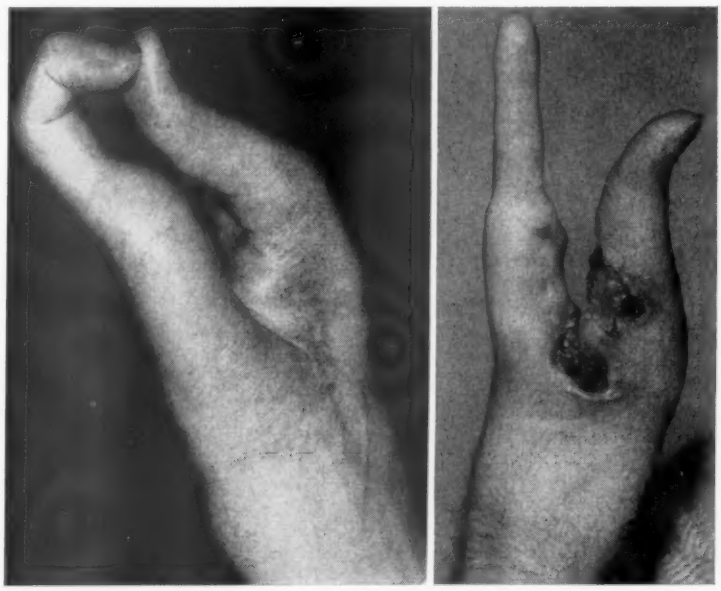
The first patient was a 67-year-old woman of Negro and Indian birth with a melanoma on the right index finger biopsied and diagnosed as melanoma by Dr. James Ewing in December, 1934 (Fig. 12, *A*). Amputation of the distal phalanx was performed elsewhere with chemical cautery. Seven years later a node appeared in the axilla which was excised elsewhere and called fibrosarcoma, but review showed metastatic melanoma with considerable spindling present. An axillary dissection revealed no residual melanoma. In October, 1946, 4 years after the axillary dissection, a subcutaneous nodule in the epitrochlear region was excised and it proved to be metastatic melanoma. Almost 4 years later a similar nodule was excised (Fig. 12, *B*). In May, 1951, an additional year later, 2 nodules appeared in the upper arm. One of these was metastatic melanoma similar to those previously removed, and the other was a separate primary melanocarcinoma (Fig. 12, *C*). The patient refused further therapy and, when last examined in August, 1952, showed no further evidence of melanoma. This was 20 years after the appearance of the primary melanoma on the finger.

The problem of multiple primary melanomas has been previously discussed by Allen,[4] Pack, Scharnagel, and Hillyer,[49] and Allen and Spitz.[5]

A second unusual patient was a 39-year-old man with a 7-year history before his admission to the Memorial Cancer Center. He had had a small nodule in the palm which had been treated by irradiation and excision. It recurred 3 years later and was excised again. It was considered to be a squamous carcinoma. Because of recurrence on admission, a disarticulation of the second, third, and fourth metacarpals was performed in September, 1947, and the defect was repaired with a full-thickness graft. At the same operative seance, preliminary excisional biopsy of axillary nodes showed metastatic melanoma, but no residual tumor was found on axillary dissection. His "lobster claw" hand was satisfactory in his work as a railway expressman (Fig. 13, *A*). A recurrence of the melanoma was found against the base of the first metacarpal bone 3 years later, as well as recurrence in the axillary scar. The patient refused any treatment then. Six months later x-ray evidence showed a solitary metastasis in the lung. Two years later a palliative interscapulothoracic amputation was performed because of the recurrence (Fig. 13, *B*). Over 2 years later a right lower lobectomy was performed for the pulmonary tumor which was thought to be an anaplastic epidermoid carcinoma but it proved to be a separate

primary melanocarcinoma of the bronchus. Numerous cutaneous lesions appeared and many of those excised proved to be independent primary melanomas. The patient survived over 14 years after appearance of the primary melanoma in the palm and 8 years after an unsuccessful axillary dissection.

Summary of End Results in Treatment of Melanoma of the Hand.—In 2 patients axillary dissection was obviated by actual excision of the axilla and contents, that is, by interscapulothoracic amputation, for bulky metastases. These patients survived 18 and 23 months, respectively. For comparative purposes, mention is made of Pack and Livingston's[50] report in 1938 of 32 axillary dissections for melanoma at various sites in the upper extremity with 4 (12.9 per cent) 5-year survivors without recurrences. Though this series is much



A. *B.*

Fig. 13.—*A*, "Lobster claw" hand of B. M. subsequent to partial amputation of hand 7 years after initial melanoma appeared in the palm. *B*, Second local recurrence in the hand just before palliative interscapulothoracic amputation 6 years after first photograph.

smaller, a 25 per cent 5-year cure rate exists for a total of 16 patients with axillary dissection and axillary ablation, that is interscapulothoracic amputations, with nodes containing metastases in 9 instances, though only one of the survivors had positive axillary nodes. Two patients survived over 5 years with persistent melanoma; one of these had a subsequent palliative interscapulothoracic amputation.

In review of the group of 29 patients with melanoma of the hands, there were 20 determinate cases, and 7 of these were alive and well over 5 years after last treatment of melanoma. One died due to a heart attack and apparently free of melanoma over 5 years after treatment, giving a 40 per cent 5-year cure rate.

SUBUNGUAL MELANOMA

One of us (G. T. P.)[45] has previously studied the problem of subungual melanoma, reporting a 21-year experience from 1917 through 1937 at the Memorial Cancer Center. Sixteen cases were reported of a total of 477 (3.4 per cent). Allen and Spitz,[5] in a later analysis of the same source material from the Memorial Hospital, found 35 (3.7 per cent) of 934 cases. In this study there were

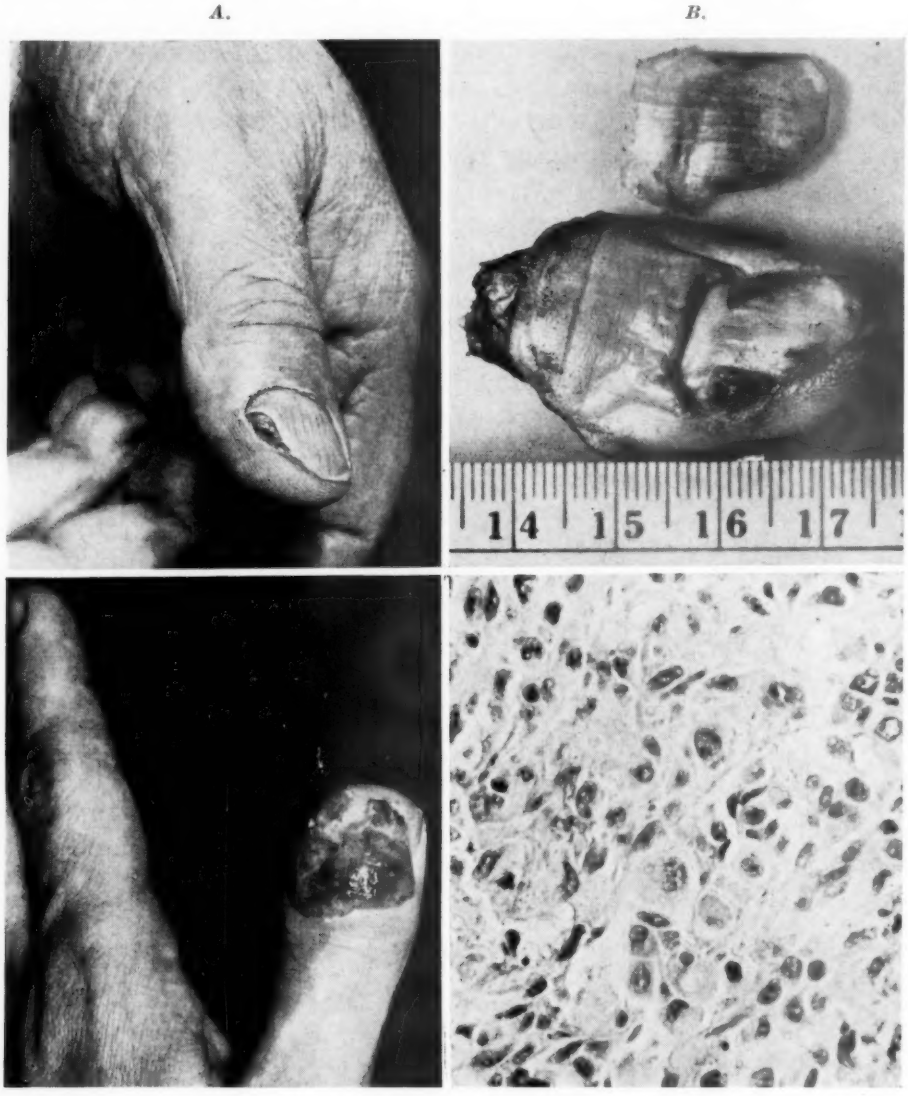


Fig. 14.—*A*, A small subungual melanoma of thumb of P. Mc., a 61-year-old man with a 3-year history of his nail "cracking" on the radial side with a subsequent growth of "proud flesh." Despite the long history the lesion is comparatively superficial in the nail bed. *B*, The distal interphalangeal joint disarticulation with the nail removed, showing a small rather superficial melanoma. The patient was without evidence of metastases 6 years later when last examined. (Private patient of Dr. Gordon McNeer.) *C*, Thumb of M. C., a 38-year-old man who had first noticed gradual separation of the nail from its bed 2 years before this. It became sensitive. The nail was cut away and never regrew. Ten months before this photograph, exuberant granulomatous-appearing tissue appeared and 4 months before this photograph it had been electrocauterized without any suspicion of the possibility that it might be a melanoma. Biopsy confirmed the diagnosis of melanoma. There has been no recurrence or metastases apparent in 7 years subsequent to metacarpophalangeal disarticulation. *D*, Photomicrograph of the subungual melanoma showing an anaplastic melanoma. This phase could be confused with a rhabdomyosarcoma (×200, ½ off reduction).

29 subungual melanomas in the group of 151 cases on the hands and feet, 13 of 29 cases in the hands (44.8 per cent), and 16 of 122 on the feet (13.1 per cent), an experience different from Meyer and Gumport's[37] dictum that the tumor was twice as common under the fingernail as under the toenail and Wright, Clark, and Milne[62] who reported 2 subungual melanomas of 54 on the feet, as opposed to 6 of a total of 8 on the hand. Affleck[3] analyzed 6 cases "beneath or about the nail" of the great toe and 3 beneath the thumbnail in a series of 317 cases.

A. *B.*

Fig. 15.—*A*, Melanotic whitlow in a 61-year-old man. Small streaks appeared around the thumbnail 5 years previously and slowly the nail turned brown before falling off. Axillary nodes also contained metastases. The patient refused interscapulothoracic amputation. A disarticulation of the thumb and axillary dissection were performed elsewhere but the patient died of diffuse metastases 16 months later. *B*, Foot of B. N., a 60-year-old man who 10 months before noticed that his great toenail had turned black. The toenail was removed and no pathologic study done, and the area remained colorless. A bleb occurred and it was cauterized, but a biopsy was not performed until 18 months of various treatments were given. This photograph shows an ulcerated, infiltrating pigmented lesion. No palpable tributary nodes were felt. There was no evidence of melanoma when last examined 12 years after metatarsophalangeal joint disarticulation.

Womack[61] observed that the nail beds of the fingers are more frequently involved than those of the toes and that nearly half of the reported cases were on the thumb. In this series 4 of 13 on the hands were on the index finger and 5 were on the thumb (Figs. 14, *A*—*D* and 15, *A*), whereas of 16 subungual melanomas on the foot the tumor was found 8 times beneath the right great toenail (Fig. 15, *B*) and 7 times beneath the left great toenail. These findings corroborate Handley's[29] much earlier observation that melanotic whitlows (a term coined by Jonathan Hutchinson and deplored by Handley) occur most frequently upon the outer side of the great toe and upon the index finger.

Of those patients with subungual melanomas of the fingers, 5 had axillary node dissections and one subsequently had the procedure performed elsewhere.

One patient had an interscapulothoracic amputation, and 6 had local treatment only. Four patients had elective node dissections, with metastases being proved twice. These patients died with melanoma. One showed phagocytized melanin only, though he died with cerebral metastases 36 months later. One patient who had shown no proof of axillary metastases died of melanoma 21 months after the dissection. The patient who had an interscapulothoracic amputation died 23 months postoperatively after a neck dissection with recurrence in the amputation scar. One patient had a therapeutic node dissection but died of metastases and local recurrences in the arm 18 months later.

Of the remaining 6 patients, 3 were indeterminate, 2 were alive and well 80 and 85 months respectively, after digital disarticulation (Fig. 14), and one died of a heart attack without apparent melanoma 50 months after amputation. Only 2 of the 13 patients are alive and well (15.4 per cent).

Four of the 16 patients (25 per cent) had 5-year cures or more and have not subsequently had further melanoma found. It is surprising that while melanoma of the hand carries a much better prognosis in general this does not hold for subungual melanoma, and the tumors beneath the nail of the toes have given a better prognosis than on the hand. The 5-year absolute survivor rate of the 29 cases was 6 (20.7 per cent). We cannot corroborate a common feeling that subungual melanoma is a less malignant tumor than those occurring in other areas of the body. This statement must take into consideration, however, a factor which we have found difficult to evaluate, namely, injudicious local maltreatment of the subungual melanoma before the nature of the tumor is recognized. For example, in an earlier series (1939), 14 of 16 subungual melanomas seen by us had had curettage of the nail bed elsewhere. Many of the patients in this present series had improper surgical trauma to these tumors before we saw them for digital amputation. It is reasonable to assume that this may have hastened or increased the tendency of the tumor to metastasize.

Of the 16 patients whose subungual melanomas were on the feet, 9 patients had groin dissections and all except one were considered therapeutic dissections. In the one considered elective, metastases were found. This patient with the elective dissection was a long-term survivor and died of other causes. Of the remainder, one was indeterminate and the others died 18, 5, 4, 13, 45, and 18 months, respectively, after dissection and one has persistent melanoma 78 months after groin dissection. One patient with a hip joint disarticulation and a retroperitoneal dissection died probably of metastases 65 months later. Of the remaining 6, 3 were long-term survivors (120, 102, and 63 months, respectively), one died 58 months after amputation of the toe and with satelletosis in the extremity, and one died 26 months postamputation. The last patient was considered at first to be a 5-year determinate controlled case, but more recently has had a node dissection for proved metastases 73 months after disarticulation of the left third toe (Fig. 1).

SUMMARY AND CONCLUSION

1. The present study is based upon an analysis of all the malignant melanomas of the hands and feet at the Memorial Cancer Center between 1935 and

1950, inclusive. In this 16-year period, 16.5 per cent of all the malignant melanomas were on the hands and feet, though these surfaces constitute only 10.5 per cent of the total body skin surface. There were 29 melanomas of the hands (3.2 per cent) and 122 malignant melanomas (13.2 per cent of the entire group) on the feet.

2. The malignant melanomas of the hands and feet occurred approximately 40 per cent in men and 60 per cent in women.

3. The patients were aware of a pre-existing mole on the foot in 24.8 per cent of all cases of malignant melanoma.

4. In 32 of the 122 melanomas of the feet (26.2 per cent), local excision only was performed, and of these the 5-year definitive cure rate was 34.4 per cent.

5. The 5-year survival rate without recurrence in the interval for all malignant melanomas of the feet was 25.3 per cent.

6. In the patients with malignant melanomas of the feet in whom metastases became evident during the course of follow-up observation, an average of 20.7 months elapsed before the metastases were detected.

7. Forty-six patients had radical groin dissections for proved metastases, and of these the 10-year definitive cure rate was 10.9 per cent.

8. The total group of all elective groin dissections without clinical evidence of metastases to nodes yielded a 5-year definitive cure rate of 33 per cent.

9. Elective groin dissections without clinical evidence of metastases but in which on microscopic study the nodes were found to contain metastases yielded a 5-year definitive cure rate of 20 per cent.

10. In elective groin dissections without clinical evidence of metastases and with groin nodes found to be negative for metastases on microscopic study, the 5-year definitive cure rate was 40 per cent.

11. Of all patients undergoing surgical treatment for malignant melanoma of the feet, 16.5 per cent subsequently had either local recurrence on the feet or widespread dissemination throughout the corresponding leg.

12. Of all the patients having groin dissection for malignant melanoma, 14 of the 56 (25 per cent) had diffuse recurrence in the extremity or in the scar of the groin dissection. Only one of these patients failed to have nodal metastases pathologically proved.

13. For patients with malignant melanomas on the feet having simultaneous nodal metastases in the groin, the 5-year definitive cure rate was only 12 per cent.

14. In a consideration of the influence of the size of the primary melanoma on the cure rate, the 5-year definitive survivals in comparison with the diameters of the primary melanomas were as follows: 1 cm., 29.4 per cent; 1 to 2 cm., 20.7 per cent; 2 to 3 cm., 22 per cent; 3 to 4 cm., 18.1 per cent; and 4 to 5 cm., 10 per cent.

15. The total salvage rate for malignant melanomas of the hand was 40 per cent, based on the 5-year definitive cure rate.

16. The 5-year definitive cure rate for malignant melanomas of the hand metastatic to the axilla was 16.6 per cent.

17. The 5-year definitive cure rate for subungual melanomas was 20.7 per cent.

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Editorial

CONDUCTED BY I. S. RAVDIN, M.D.

Intestinal Obstruction

Intestinal obstruction, a major problem for generations of surgeons, has seen more rapidly changing concepts as to pathogenesis and treatment during the last 50 years than almost any other clinical entity. Many ideas, both as to pathogenesis and treatment, have arisen along the way, and newer therapeutic measures, one after another, have been enthusiastically adopted, all too often injudiciously applied, and then discarded in favor of others still newer. Even today we find far too many surgeons advocating but one method or another for the treatment of intestinal obstruction, rather than a wise selection of one or more of the available techniques, according to the needs of the individual patient.

The cause of death in strangulating obstruction associated with necrosis and perforation of the intestinal wall is clear. Less easy to understand has been the rapid deterioration of many of these patients, even before perforation occurs. Recent investigations have shed additional light, but the picture is still not entirely clear. The lethal factors in nonstrangulating obstruction and the requisite therapy for their elimination have been much clarified in recent years. It has been well shown that distention, itself, will produce the entire picture of obstruction in experimental animals, even though the distended intestine be kept free of retained small intestinal content. Administration of fluids (including blood) and correction of electrolyte deficiencies and acid-base imbalance will strikingly better the condition of both obstructed animals and patients. Space does not permit the enumeration of additional findings refuting the once universally accepted idea of toxic absorption as the chief lethal factor in nonstrangulating obstruction. Suffice it to say that physiologic derangement rather than toxic absorption is now generally accepted as the basis for the disorders produced by intestinal obstruction.

Widespread administration of electrolytes and blood to these patients brought the first significant reductions in the shocking mortality rate which prevailed at the turn of the century. Nonoperative decompressive techniques were a further development in the search for a more effective therapeutic armamentarium. Wangensteen and others, with gastroduodenal suction drainage, achieved significantly lower mortality rates than any previously reported. Development by Miller and Abbott of a satisfactory technique for intestinal intubation and its application to intestinal obstruction by Abbott and Johnston were further steps toward effective nonoperative decompressive measures. Clinics utilizing a proper combination of these methods with operative treatment where

indicated, the patients being supported with electrolytes and blood, achieved for the first time mortality rates far below those of only a few years back. Nevertheless, too many patients still die from intestinal obstruction. Earlier diagnosis and judicious choice of treatment methods should do much to further improve the picture.

There need be no confusion as to the proper therapeutic approach, despite the many techniques now available. A few basic principles might well be enumerated:

1. If strangulation can be diagnosed or is even seriously suspected, there can be no justification for delaying emergency operative exploration. (Administration of blood and electrolytes is imperative during the hour or more that is required for setting up the operating room.) The hazard of strangulation is so great that delay for even a few hours of gastrointestinal suction may be disastrous.

2. Immediate operation is also in order if the diagnosis of intestinal obstruction is reasonably clear and the patient has not yet developed distention even though strangulation is not suspected. In the absence of distention sufficient to cause derangement of the patient's physiologic processes, there can be no possible indication or need for preoperative intestinal suction drainage which has as its object the relief of distention.

3. If the patient, as a result of distention, has developed the characteristic picture of fluid loss and electrolyte imbalance and if strangulation is not suspected, he needs preoperative decompression. This will permit his restoration to good physical condition for elective release of the obstructing agent. One should not persist unduly long, however, in his efforts to pass the tube beyond the pylorus. If the patient is not progressively improving during intubation or if it is not possible to pass the tube into the duodenum within 12 to 24 hours, one is not justified in further prolonging the attempts. The patient should be explored for mechanical release of the obstructing agent.

4. If the patient be suffering from adynamic ileus, gastrointestinal suction is by far the most effective means of treatment. Peristalsis-stimulating drugs have been disappointing and are of dubious value. The only other alternative is enterostomy, an unsatisfactory treatment at best, which commonly drains only a loop or two, and that poorly, and subjects the patient to additional operative trauma, which will further aggravate the ileus. (It should be borne in mind that since 70 per cent of the distending gases are swallowed air, gastric suction is the most effective means for preventing distention. Duodenal or intestinal suction drainage, however, will be vastly more effective in removing distending gases once they have accumulated.)

5. Finally, gastrointestinal suction methods are never justified as the primary approach to the treatment of colonic obstruction. There is no substitute for proximal colostomy in the management of obstruction of the large intestine.

We must never let our enthusiasm for new techniques, however valuable, lead us into slavish insistence that any one of them is *the* method of choice for all cases. Careful diagnostic studies should result in selection of the particular treatment methods indicated for each individual patient. Only thus can we hope to preserve the lives of many more patients suffering from intestinal obstruction.

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Appraisal of Progress in Surgical Therapy

CONDUCTED BY WARREN H. COLE, M.D.

THE SURGICAL TREATMENT OF LUNG TUMORS. PNEUMONECTOMY OR LOBECTOMY?

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SHOULD a lobectomy or pneumonectomy be used to treat a malignant tumor of the lung? This question is not one to be easily answered. In the past, emphatic statements have been made that pneumonectomy is the procedure of choice if a bronchogenic carcinoma is treated with the hope for cure of the disease. In more recent years evidence has accumulated that lobectomy is the operation of choice not only in the poorer risk patient but in cases where the tumor is well localized to the involved lobe and a cure is to be hoped for.

Ochsner[5] in 1954 reported his experience with 1,457 cases of bronchogenic carcinoma. In this series, surgical exploration was carried out in 49.6 per cent of the patients and a pulmonary resection (mostly pneumonectomy) was performed in 469 patients (32.1 per cent). In his article Ochsner estimated that 73 per cent of the patients in whom pulmonary resection was done had "palliative" resections. "Palliation" in this sense indicated extension of the tumor outside the lung. Conversely only 27 per cent of the cases resected had tumor confined to the lung. Obviously less than 27 per cent had lesions wherein a lobectomy could have been considered as a curative operation. The operative mortality rate in those patients resected was 19 per cent. In his report Ochsner wrote: "There is no question but that some patients with bronchogenic carcinoma can be cured by the removal of a lobe, but it is strongly believed that many more patients would be cured if a radical operation, consisting of a pneumonectomy with an en bloc excision of all the mediastinal nodes, were done. Indeed, it is believed that the only indication for lobectomy for bronchogenic carcinoma is pre-operative evidence obtained from careful pulmonary function studies that a pneumonectomy could not be tolerated."

An important report demonstrating that a lobectomy had some merit in the curative treatment of bronchogenic carcinoma was made by Churchill[4] in 1950. Churchill stated that the five-year survival rate for bronchogenic carcinoma treated surgically at the Massachusetts General Hospital was 12 per cent in those patients treated by pneumonectomy and 19 per cent in those patients

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treated by lobectomy. It was pointed out that these two groups of patients could not be considered comparable. The patients treated by lobectomy fell into three categories: (1) the tumor was less extensive and confined to a lobe, (2) the patients were thought to be less good surgical risks who would tolerate a lobectomy better than a pneumonectomy, or (3) a lobectomy was performed for palliative reasons since known tumor had to be left. The increased operative mortality of a pneumonectomy as compared to a lobectomy probably helped to decrease the survival of patients with pneumonectomy in comparison to patients with lobectomy. Churchill reported that the operative mortality rate for pneumonectomy performed in the years 1933 to 1950 was 22.8 per cent. Whereas the operative mortality rate for lobectomy carried out in the years 1930 to 1950 was 14 per cent. Churchill reported that there was only one operative death in 27 pneumonectomies (3.7 per cent) and one operative death in 26 lobectomies (3.8 per cent) operated upon in the interval 1948 to 1949. This low mortality rate for pneumonectomy is not universally true and varies considerably with the type of cases operated upon. Most authors have found increased mortality rates in pneumonectomy operations compared to lobectomy operations. The disability to the patient is more marked if a pneumonectomy is done. This is particularly true if it is a right-sided pneumonectomy since this is the larger lung.

A more recent study by Overholt[6] is illustrative of the tendency to consider lobectomy as an operation of choice in the cure of some bronchogenic carcinomas. Overholt wrote: "In our clinics as the years have advanced, we have extended the completeness of lymph structure removal and have been more conservative of ipsilateral pulmonary lobes." In his series Overholt reported 733 cases of bronchogenic carcinoma of which 261 patients (37 per cent) were resected. There were 55 patients who survived five years or more. Pneumonectomy with mediastinal lymph node dissection was carried out in 51 of the patients surviving for five years or more. However, Overholt further reported that the three-year survival of 45 patients treated by lobectomy paralleled the three-year survival rate of patients treated by pneumonectomy in the same era.

The increased tendency to use lobectomy as the procedure of choice in the cure of some bronchogenic carcinomas was elaborated by Robinson, Jones, and Meyer[7] in 1956. These authors reported 216 patients with bronchogenic carcinoma operated upon between 1950 and 1955. In this group of patients 72 pneumonectomies and 49 lobectomies were performed. It was pointed out, however, that the percentage of resections consisting of lobectomy rose from 30.4 per cent in 1950 to 61.5 per cent in 1955. These authors also pointed out that the three-year survival rate of patients with bronchogenic carcinoma gave figures very similar to the five-year survival statistics. Accordingly, they felt that there was probable significance to the fact that their three-year survival rate was 71.4 per cent in patients treated with lobectomy and 16.3 per cent in those patients treated by pneumonectomy. In this same paper Robinson

and associates summarized a poll they had taken of the members of the American Association for Thoracic Surgery. The purpose of the poll was to determine the current feelings of the thoracic surgeons of America concerning the role of lobectomy in the treatment of bronchogenic carcinoma. The questionnaire was answered by 318 members. The following question concerning the use of lobectomy was asked in the poll: "Given the indications which you consider would make the operation one of choice, is it your experience that it offers the patient as good a chance for long-term survival as does pneumonectomy?" One hundred and seventy-three thoracic surgeons (56.9 per cent) answered "Yes."

Bronchogenic carcinoma in consecutive unselected cases has continued to have a most unfavorable prognosis. In the consecutive unselected series, 10 per cent will survive five or more years at best. Two lines of attack may be used to better these figures. One approach is to make the procedure more "radical" in the hope that the disease can be more completely removed.

Cahan, Watson, and Pool[3] in 1951 described their technique of "radical" pneumonectomy. In a radical pneumonectomy an attempt is made to remove all the obtainable lymph nodes en bloc with the diseased lung. This operation is in contrast to that of "simple" pneumonectomy where the lung is removed with no attempt to dissect out the lymph nodes of the mediastinum. From the surgical literature it is difficult to ascertain how many pneumonectomies are radical or what the term radical really means. The right-sided pneumonectomy lends itself to an en bloc excision of the lymph nodes much more readily than does the left-sided pneumonectomy where the aortic arch interferes with the completeness of the mediastinal dissection. Perhaps the number of lymph nodes found in the resected specimen could be used as a criterion of "radical." A recent study to count and map the average number of mediastinal lymph nodes was made in our laboratory. The mediastinum at autopsy was removed en bloc and cleared by the Gilchrist modification of the Spalteholz technique. Approximately 70 lymph nodes were found in the mediastinum. Approximately 50 lymph nodes were found adjacent to the trachea and main bronchi. The identification of 50 lymph nodes in the surgical specimen would seem to indicate a radical pneumonectomy.

The other main approach to improve the survival figures in the treatment of bronchogenic carcinoma is the diagnosis in the early or silent phase of the disease when the tumor is confined to a lobe of the lung. The education of the lay public and the physicians to obtain frequent routine roentgenograms of the chest and to diagnose all pulmonary lesions promptly should result in surgical excision of bronchogenic carcinomas at an earlier date. Thoracic surgeons have found that the majority of asymptomatic bronchogenic carcinomas can be resected, whereas the majority of symptomatic carcinomas cannot. Much remains to be done from an educational viewpoint; Ochsner has stressed that the delay caused by the patient is approximately 2 to 3 months but the delay caused by the physician has risen to approximately 6 months in this antibiotic era.

A factor that should tend to improve the survival rate in patients treated surgically is improvement in surgical techniques. Better anesthesia, the use of blood transfusions, the availability of antibiotics, and better surgical techniques give lower operative mortality rates which tend to augment survival rates. This tendency is somewhat balanced by the surgeon's willingness to accept poorer risks for surgical treatment. Operative mortality will increase with advancing age. Yet more aged patients are seen by the surgeon. Our oldest patient who underwent successful pneumonectomy for bronchogenic carcinoma was 79 years of age. The operative mortality will increase if the maximum breathing capacity is below 40 per cent of predicted normal values. Yet if nonfunctioning lung tissue is removed and if the surgeon is prepared to assist the patient's respiration mechanically in the immediate postoperative period, very poor risk patients can be carried through a pulmonary resection. Our patient in the worst functional condition had a maximum breathing capacity of 19 per cent of predicted normal. He had a successful right upper lobectomy for a carcinoma which was destroying this lobe but was without obvious spread outside the lobe. His respiration was mechanically assisted for one week postoperatively. Some months later his pulmonary function tests approximated his preoperative values.

A technique in the treatment of carcinoma that is seldom successfully applicable to lung carcinomas is that of the "second look" operation proposed by Wangensteen. In 1953 we saw a patient who had had a right pneumonectomy 18 months before for an epidermoid carcinoma in the right upper lobe. He re-entered the hospital with hemoptysis and on bronchoscopic biopsy was found to have an epidermoid carcinoma in the stump of the right main bronchus. He was re-explored through the right thorax. The bronchial stump, the coryna, and part of the lateral tracheal wall were excised. No tumor-bearing lymph nodes were found. The defect was repaired with steel wire mesh and fascia lata. He is now alive and well more than four years after his operation for recurrent bronchogenic carcinoma. Our attempts to carry out "second look" operations after left-sided pneumonectomy have not been successful. The presence of the aortic arch on the left had made dissection of tumor in the vicinity of the bronchial stump hazardous.

Metastatic malignant tumors in the lung may present difficult problems. Solitary metastatic tumors in the lung should be resected if the primary tumor is controlled. It is particularly important to realize that what appears to be a solitary metastasis may in fact be a new primary tumor. Multiple primary tumors occurring in various areas of the body are not rare.

Multiple metastatic tumors in the lungs are very unfavorable but not entirely hopeless. It is probably slightly better if the multiple metastases appear to be confined to one lung. If the primary tumor is controlled, if the patient is a good surgical risk, and if the lesions are so located as to make excision feasible, surgery may be offered to the patient.

Less than lobectomy, or lobectomy, is the operation of choice in treating metastatic malignancies. At times a patient will be seen with unilateral metastases wherein the lung or hilum is involved in a fashion necessitating a pneumonectomy.

SUMMARY

Pneumonectomy has been the preferable treatment of bronchogenic carcinoma where a cure is to be hoped for. However, there has been a recent growing tendency to use lobectomy as a curative procedure for certain bronchogenic carcinomas. The reported similar survival figures for lobectomy and pneumonectomy plus the lower mortality and morbidity figures for lobectomy have influenced this tendency. At the present time the following statements are suggested.

Lobectomy should be definitely indicated when (1) the patient is a poor surgical risk; (2) the patient cannot be cured, but it seems that lobectomy will afford palliation equal to that of a pneumonectomy; (3) the indeterminate lesion which might or might not be a tumor but is confined to one lobe.

The probable indication for a lobectomy is in the case of an early, well-circumscribed tumor, preferably asymptomatic and peripheral, especially if the tumor is in the right upper lobe where little more margin would be obtained by pneumonectomy.

Pneumonectomy is definitely indicated (1) where the tumor has extended outside the lung but the extensions can be removed by a "radical" pneumonectomy; (2) where the tumor is apparently confined to the lung but involves more than one lobe; (3) where the tumor is confined to one lobe, but the lobe is extensively involved, most especially if the tumor is in a left lobe where more margin could be obtained with a pneumonectomy than a lobectomy; (4) for the palliation of cancers where a lobectomy will be technically impossible or give less palliation.

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Book Reviews

CONDUCTED BY J. GARROTT ALLEN, M.D.

A Frontal Section Anatomy of the Head and Neck. Otto Frederic Kampmeier, Arthur R. Cooper, and Thomas S. Jones. Urbana, Ill., 1957, University of Illinois Press. 5 ills., 20 frontal sections. $15.00.

For anyone who has had frequent occasion to refer in neurosurgical work to the great William Macewen's *Atlas of Headsections* (1893), the appearance of this new Atlas will be welcome indeed. Macewen's Atlas, produced in the midst of a tremendously busy surgical life, was a classic. The present volume merits a similar superlative; in fact, in its scope, number of plates, detailed identification, and beauty of production, it far surpasses Macewen's Atlas.

I believe that thanks are due not only to the authors, but also to the University of Illinois Research Board for its wisdom in authorizing publication, and to the past and present Directors of the University of Illinois Press, and their associates, for this beautiful piece of bookmaking.

The senior author has included an interesting review of the historical development of cross sectional studies in anatomy.

This book can be recommended unreservedly to anyone involved in anatomic or surgical studies of the head and neck.

—Joseph P. Evans

Essentials of Clinical Proctology. Manuel G. Spiesman and Louis Malow. Ed. 3, New York, 1957, Grune and Stratton, Inc. Pp. 316. $8.75.

The third edition of this widely read book on diseases of the anus, rectum, and colon again presents only the essentials of the subject of proctology. Each disease entity is discussed briefly from the standpoint of anatomy, etiology, pathology, symptoms, differential diagnosis, and treatment. Step by step descriptions of techniques in proctoscopy, sigmoidoscopy, anesthesia, and operative procedures are clearly explained and well illustrated. Treatment and techniques are presented in a rather dogmatic manner including only those that the authors consider the most satisfactory without presenting other currently used prescriptions and techniques.

Among new chapters added to this edition are ones discussing the injection treatment of hemorrhoids, pediatric proctology, ulcerative colitis, hydradenitis suppurative, coccygodynia, and proctalgia fugax, amebiasis and its relationship to pruritis ani, and a new histopathologic classification of polyps and their treatment.

Twenty pages are devoted to a dissertation on the pecten band, pectenosis, and pectenotomy—an understanding of which the authors feel is important in arriving at the proper diagnosis and treatment of proctologic diseases.

The chapter on the subject of pruritis ani has been revised with a discussion of the use of newer drugs and the "clover leaf" operation for the intractable condition.

The book, although far from an exhaustive treatise on the subject of proctology, should be found useful by the medical student, resident, and practitioner as a practical reference.

—Robert W. Harrison

Atlas of Clinical Endocrinology. H. Lisser and R. F. Escamilla. St. Louis, 1957, The C. V. Mosby Company. Price $18.75.

The authors state in their preface "There are many excellent textbooks on the endocrinopathies. Some of them are elaborately illustrated, but the overwhelming emphasis in most instances has been focused on the written word. . . . The primary purpose of this work . . . is to offer a concise and largely visual presentation of endocrine disorders in atlas format, and an abbreviated text which contains all essentials for adequate diagnosis and treatment even to details of dosage." This is a rather accurate thumbnail sketch of the *Atlas of Clinical Endocrinology.* There is a profusion of photographs which are on the whole elegantly presented, many of them from the collection of the authors, many others which would be familiar to long-time readers of endocrine texts. The coverage is reasonably comprehensive ranging from the usual endocrinopathies through a variety of what have been termed miscellaneous disorders such as mongolism, achondroplasia, gargoylism, hyperostosis frontalis interna, and so forth. Diabetes mellitus has been deliberately omitted because it did not lend itself to photographic presentation. The only other omission I noted is that of pseudohypoparathyroidism which is only referred to in a sentence.

The illustrations are excellent throughout although in some circumstances the choice of cases might be criticized on the basis of being extreme. More detailed presentation of less grotesque cases might, from some points of view, be useful. This was particularly true with respect to the section on goiters.

The text is brief indeed. With a dynamic subject such as clinical endocrinology it is difficult to be so brief and yet avoid the criticism of dogmatism and/or naïveté. These pitfalls have not been avoided throughout. As the sole text for a medical student it would accordingly seem inadequate. However, as a supplement for some acceptable endocrine text "focused on the written word" this atlas is probably a worthwhile addition. To the practicing clinician to whom it is also directed it may be of value as a quick reference text. The teacher of endocrinology might also find it useful because of the illustrations. Unfortunately the references given at the end of each section of the text are inadeqquate. The limitations of the short text would be more effectively overcome if more detailed reference material had been supplied.

—Richard L. Landau

The Early Diagnosis and Treatment of Acoustic Nerve Tumors. J. Lawrence Pool and Arthur A. Pava. American Lecture Series No. 303. Springfield, 1957, Charles C Thomas. 161 pp., 33 ills. $5.50.

It was Harvey Cushing in his monograph with Louise Eisenhardt on *Meningiomas* who pointed out that the future further advances in neurologic surgery would come from careful exploitation of the biologic behavior and surgical handling of individual tumors. The present volume amply confirms this contention and supplements beautifully Cushing's own classic, *Tumors of the Nervus Acusticus* (1917).

The authors briefly review history, terminology, histogenesis, and pathology, in successive chapters. Subsequently, they emphasize the great importance of the chronological development of symptoms, stressing the relative unimportance of tinnitus as a presenting symptom in contrast to the generally held opinion, and pointing out the value of objective signs of trigeminal impairment and of reduced labyrinthine function.

They add nothing of significance in discussing diagnostic procedures, and, in fact, have utilized air studies infrequently, despite the apparent safety and value of the procedure as established by the Swedes.

It is the sections dealing with the operative handling and results that are most challenging, largely because the senior author, director of one of the few remaining large neurosurgical centers in the country, has undertaken himself the responsibility of sugical management of this difficult lesion and has demonstated again, as did Cushing and

others subsequently, that nothing substitutes for accumulated technical skill. Pool's personal mortality figures were 16.2 per cent in 42 cases operated upon between 1948 and 1956, by no means a poor figure: however, in 29 cases operated upon in the last 4 years of that 8-year period, he reduced the figure to 6.8 per cent. Significantly, the end results are better when total removal of the tumor is accomplished and the threat of recurrence is removed.

These observations indicate clearly that a young surgeon is "brought up" to tackle these difficult lesions. They also cause one to wonder whether the lesions should be attacked by an individual surgeon so rarely as to be for him only an occasional surgical exercise.

Though there is little that is new, other than superb results, this is a book that should certainly be read carefully by anyone called to deal with such lesions.

—Joseph P. Evans

Calendar of Surgical Meetings

1957

AMERICAN MEDICAL ASSOCIATION. *Clinical Meeting,* Dec. 3-6, 1957, Auditorium, Philadelphia, Pa. Deadline for papers, July 15, 1957. Secretary, American Medical Association, George F. Lull, M.D.; Secretary, Council on Scientific Assembly, Thomas G. Hull, M.D.; address of both, 535 North Dearborn Street, Chicago 10, Ill.

SOUTHERN SURGICAL ASSOCIATION. *Annual Meeting,* Dec. 10-12, 1957, Greenbrier Hotel, White Sulphur Springs, W. Va. Secretary, George G. Finney, M.D., 2947 St. Paul St., Baltimore 18, Md. Guests are invited by the Secretary at the request of the Membership.

1958

AMERICAN ASSOCIATION OF GENITO-URINARY SURGEONS. *Annual Meeting,* April 23-25, 1958. Edgewater Gulf Hotel, Edgewater Park, Miss. Secretary-Treasurer, Dr. Wm. J. Engel, 2020 East 93rd Street, Cleveland, Ohio.

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY. *Annual Meeting,* May 16-18, 1958. Statler Hotel, Boston, Mass. Secretary, Dr. H. T. Langston, 1919 West Taylor Street, Chicago 12, Ill.

AMERICAN GYNECOLOGICAL SOCIETY. *Annual Meeting,* May 19-21, 1958, The Grove Park, Asheville, N. C. Secretary, Dr. Andrew A. Marchetti, Georgetown University Hospital, Washington 7, D. C.

AMERICAN SURGICAL ASSOCIATION. *Annual Meeting,* April 16-18, 1958, Waldorf-Astoria, New York, N. Y. Secretary, Dr. William A. Altemeier, Cincinnati General Hospital, Cincinnati, Ohio.

AMERICAN UROLOGICAL ASSOCIATION. *Annual Meeting,* April 28–May 1, 1958, Roosevelt Hotel, New Orleans, La. Deadline for submission of papers Dec. 1, 1957. Secretary, Dr. Samuel L. Raines, 188 South Bellevue Boulevard, Memphis 4, Tenn.

THE CENTRAL SURGICAL ASSOCIATION. *Annual Meeting,* Feb. 20-22, 1958. Columbus, Ohio. Deadline for submission of abstracts, Nov. 15, 1957. Secretary, Dr. Charles D. Branch, 1002 N. North Street, Peoria, Ill.

THE HARVEY CUSHING SOCIETY. *Annual Meeting,* April 21-23, 1958, The Mayflower, Washington, D. C. Secretary, Dr. David L. Reeves, 316 West Junipero Street, Santa Barbara, Calif.

INTERNATIONAL UNION AGAINST CANCER. *Seventh International Cancer Congress,* July 6-12, 1958, London, England. Papers, dealing with new and unpublished work, must be submitted with abstract (not over 200 words) before October, 1957. Secretary General, Harold Dorn, National Institute of Health, Bethesda 14, Md.

NEW ENGLAND SURGICAL SOCIETY. *Annual Meeting,* Oct. 3 and 4, 1958, Mount Washington Hotel, Burton Woods, N. H. Deadline for submission of papers, June 1, 1958. Secretary, Dr. Richard Warren, Peter Bent Brigham Hospital, 721 Huntington Avenue, Boston 15, Mass.

PACIFIC COAST SURGICAL ASSOCIATION. *Annual Meeting,* Feb. 3-5, 1958, Biltmore, Santa Barbara, Calif. Secretary, Dr. Carlton Mathewson, Stanford University Hospital, San Francisco, Calif.

SOCIETY OF UNIVERSITY SURGEONS. *Annual Meeting,* Feb. 13-15, 1958, Boston, Mass. Deadline for submission of abstracts three months prior to meeting. Secretary, Dr. James D. Hardy, University of Mississippi Medical Center, Jackson, Miss.

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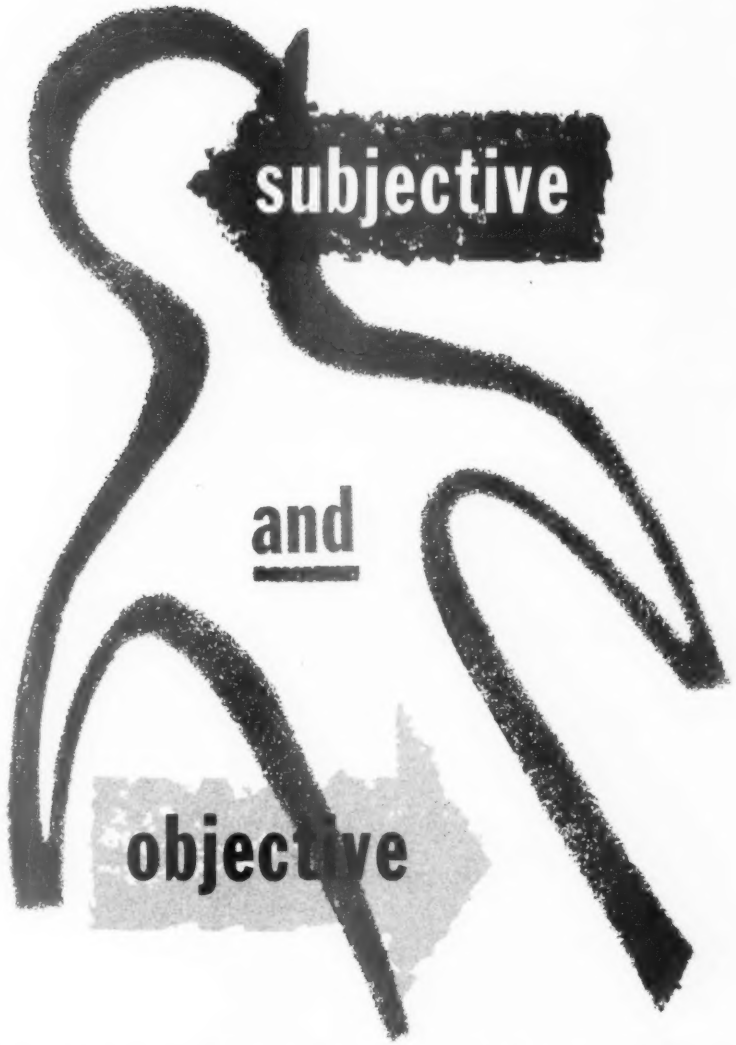

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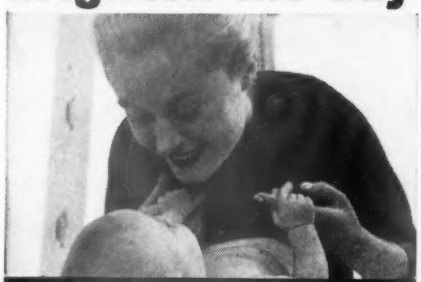

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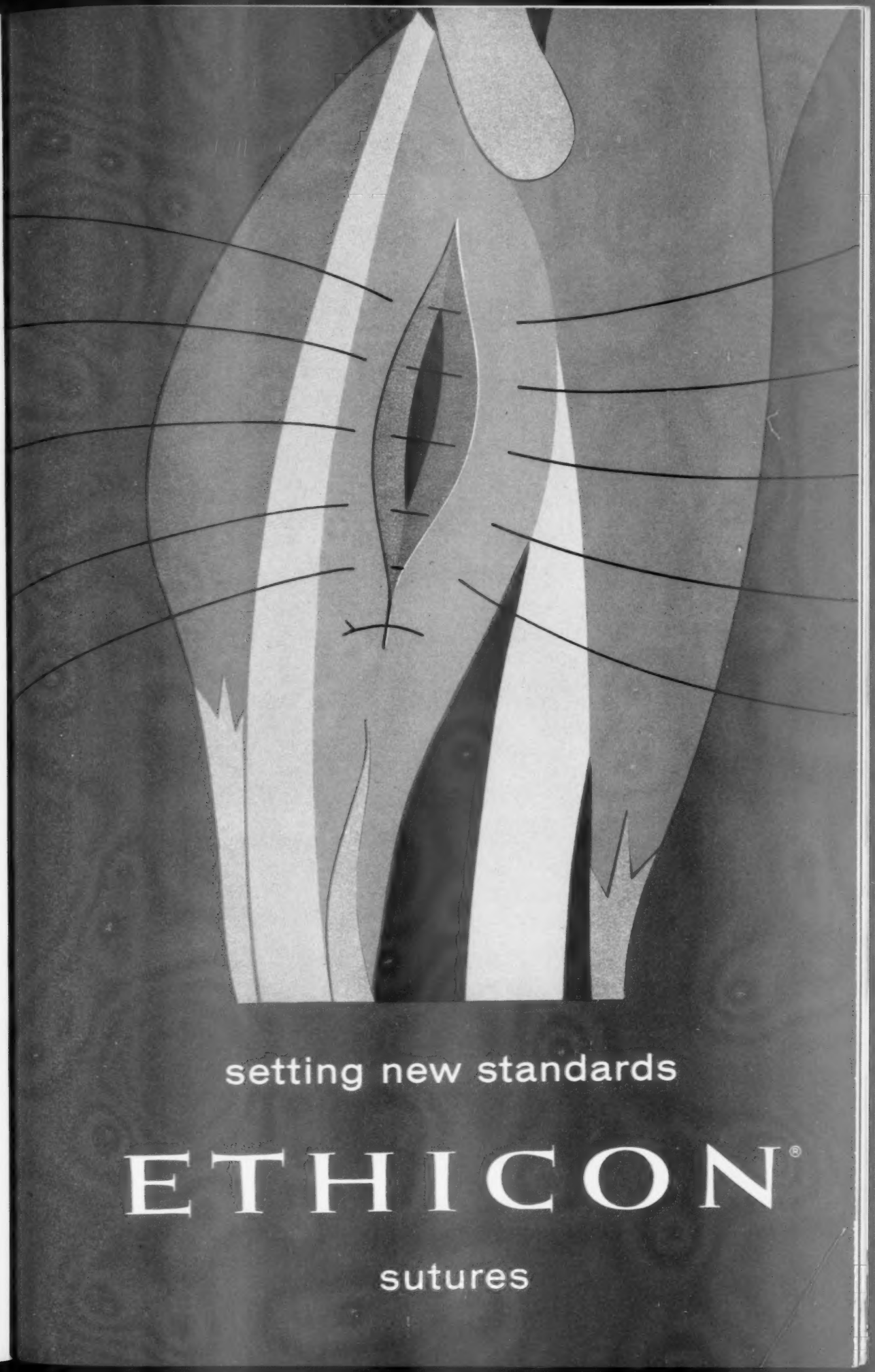
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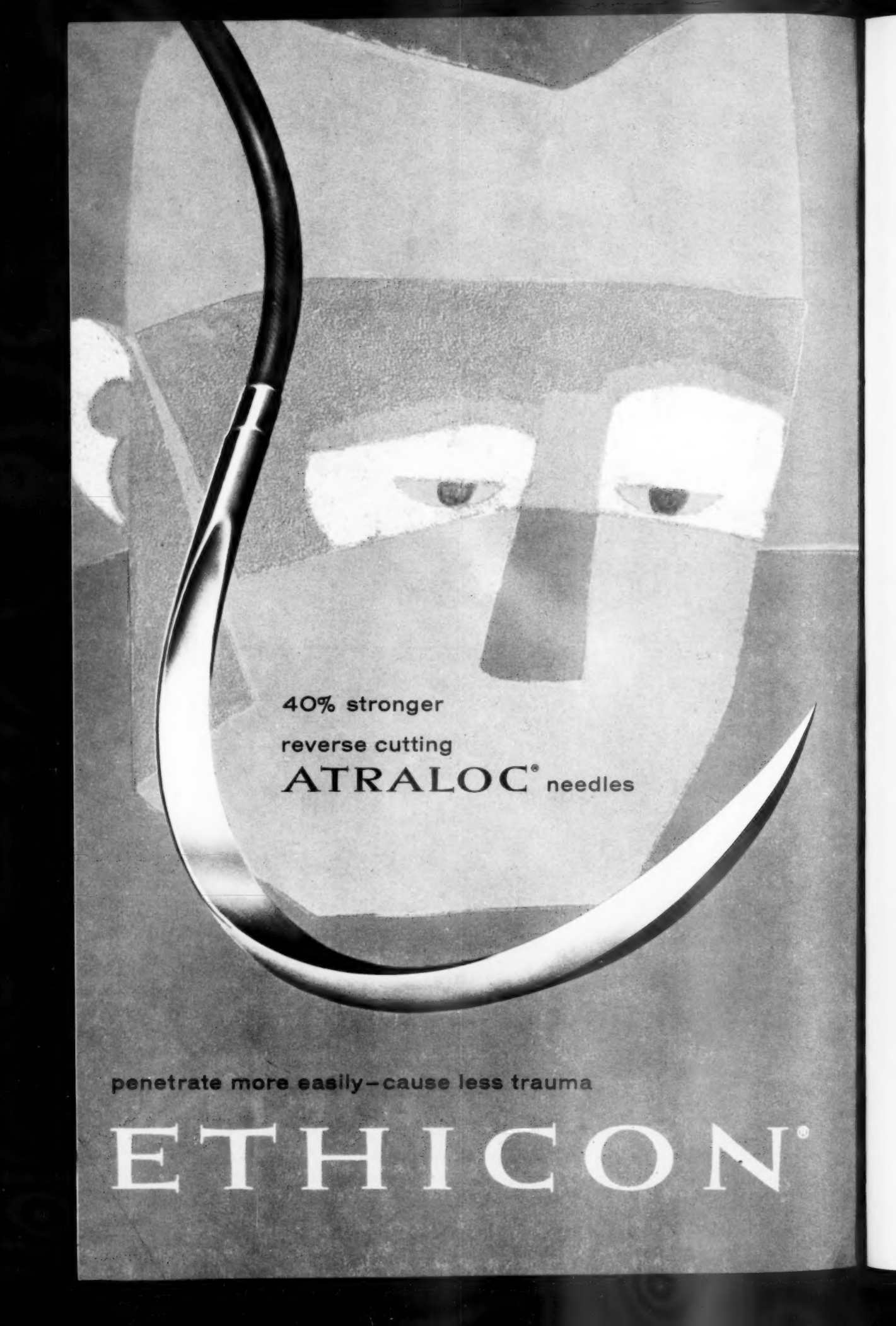
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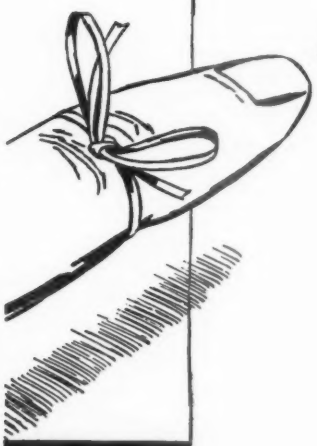

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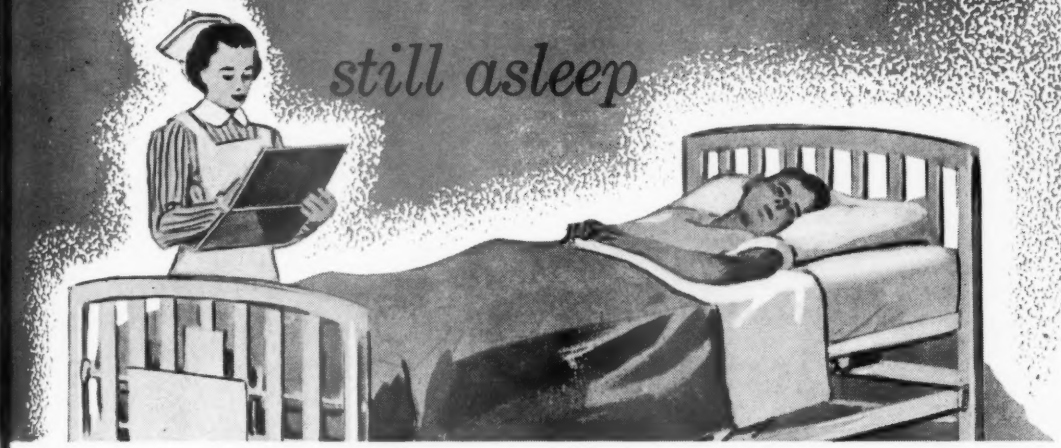

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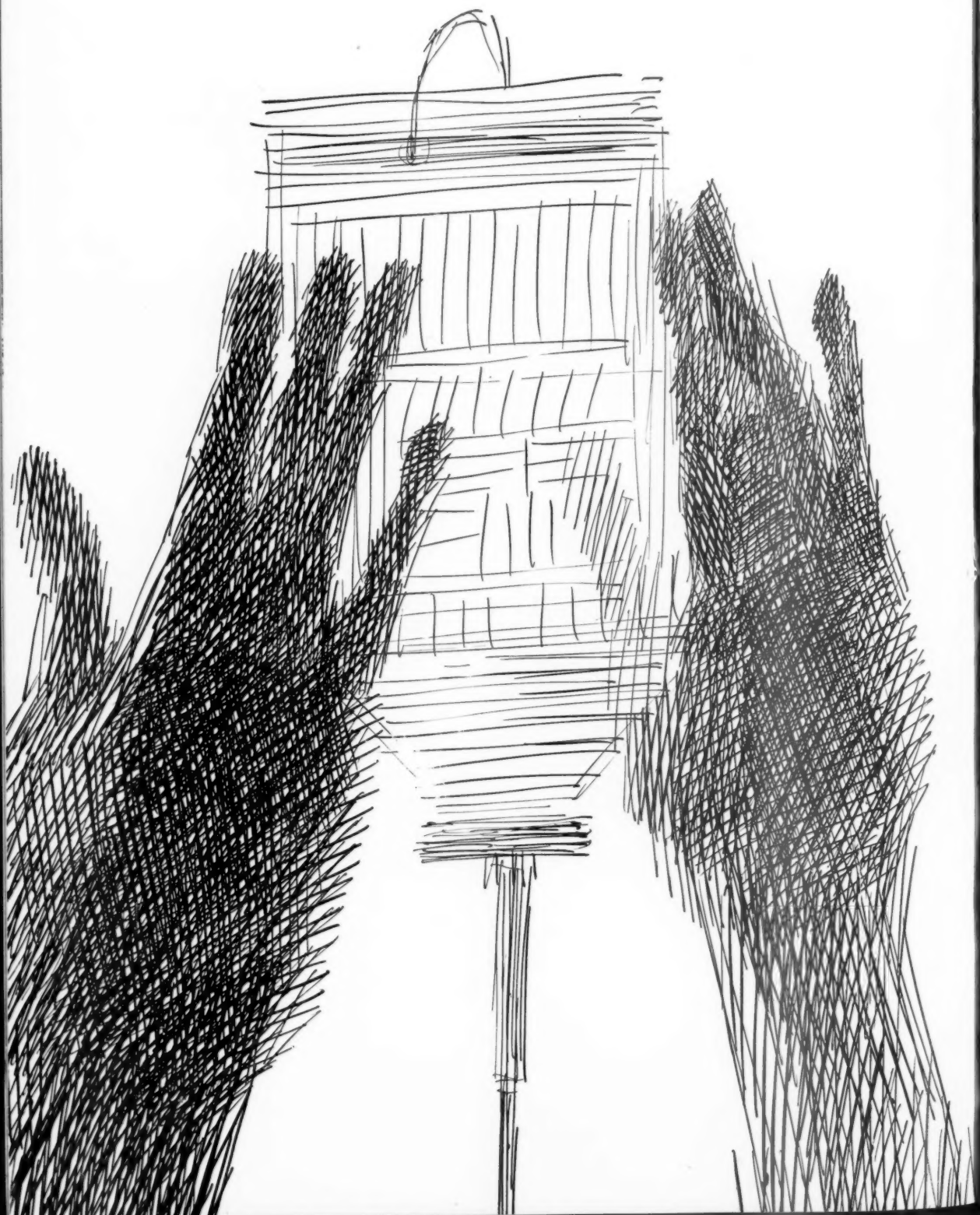

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by

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1. Riddle, A.C., Jr.: Adrenosem Salicylate: A Systemic Hemostat, Oral Surg., Oral Med., Oral Path. 6:617 (June, 1955).

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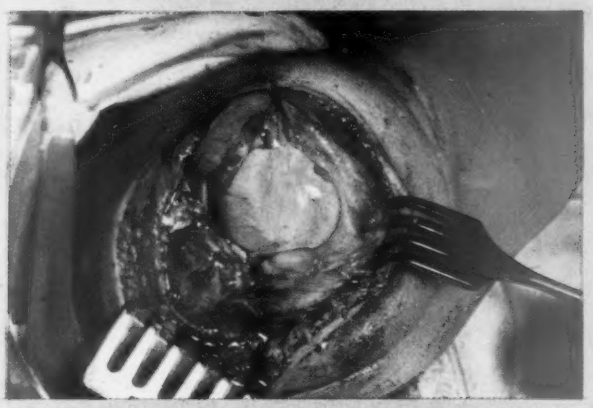
SHOULDER ARTHRODESIS:
1. Radiograph (A-P view). Shoulder prior to surgery. Note atrophy of muscle and soft tissue, and relaxation of joint.

2. Skin draped. Saber incision outlined at acromion.



3. Acromion denuded of cartilage. Shoulder capsule opened.

4. Acromial process of the scapula osteotomized.
Turn page for other color illustrations of surgery.

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and so easy—*to illustrate significant cases with radiographs and photographs . . .*

Take the shoulder arthrodesis shown here.

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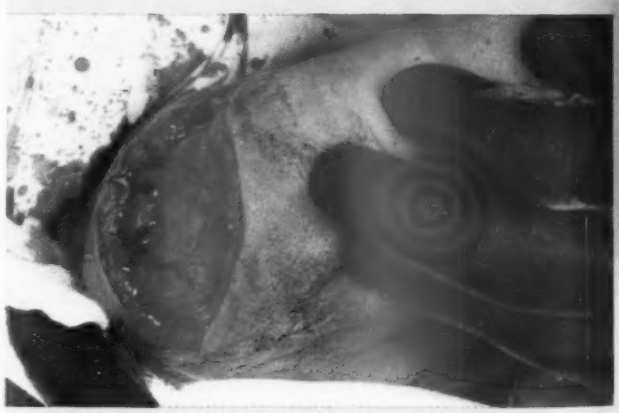
5. In succession: Flap is osteotomized in head of humerus; cartilage is denuded from head of humerus, which is joined to acromial process.

6. Acromial process now in head of humerus.



7. Cuff of shoulder capsule reefed to scapula.

8. Wound closed and prefitted cast applied.

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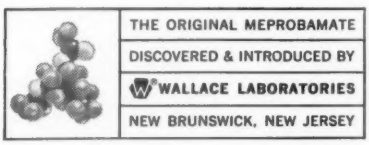

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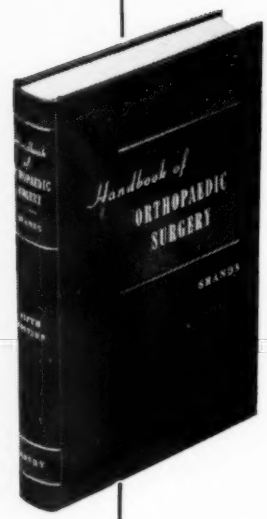

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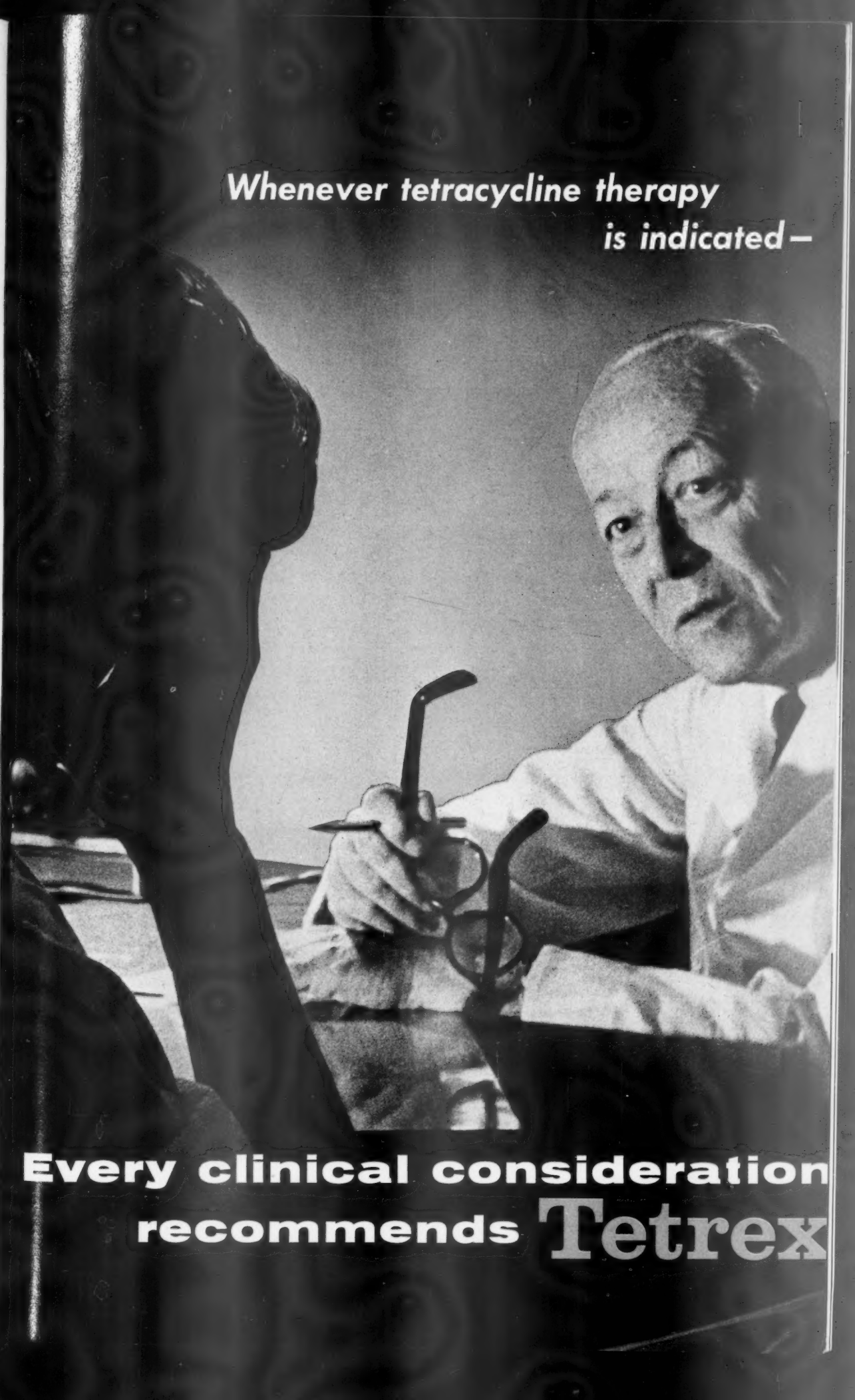
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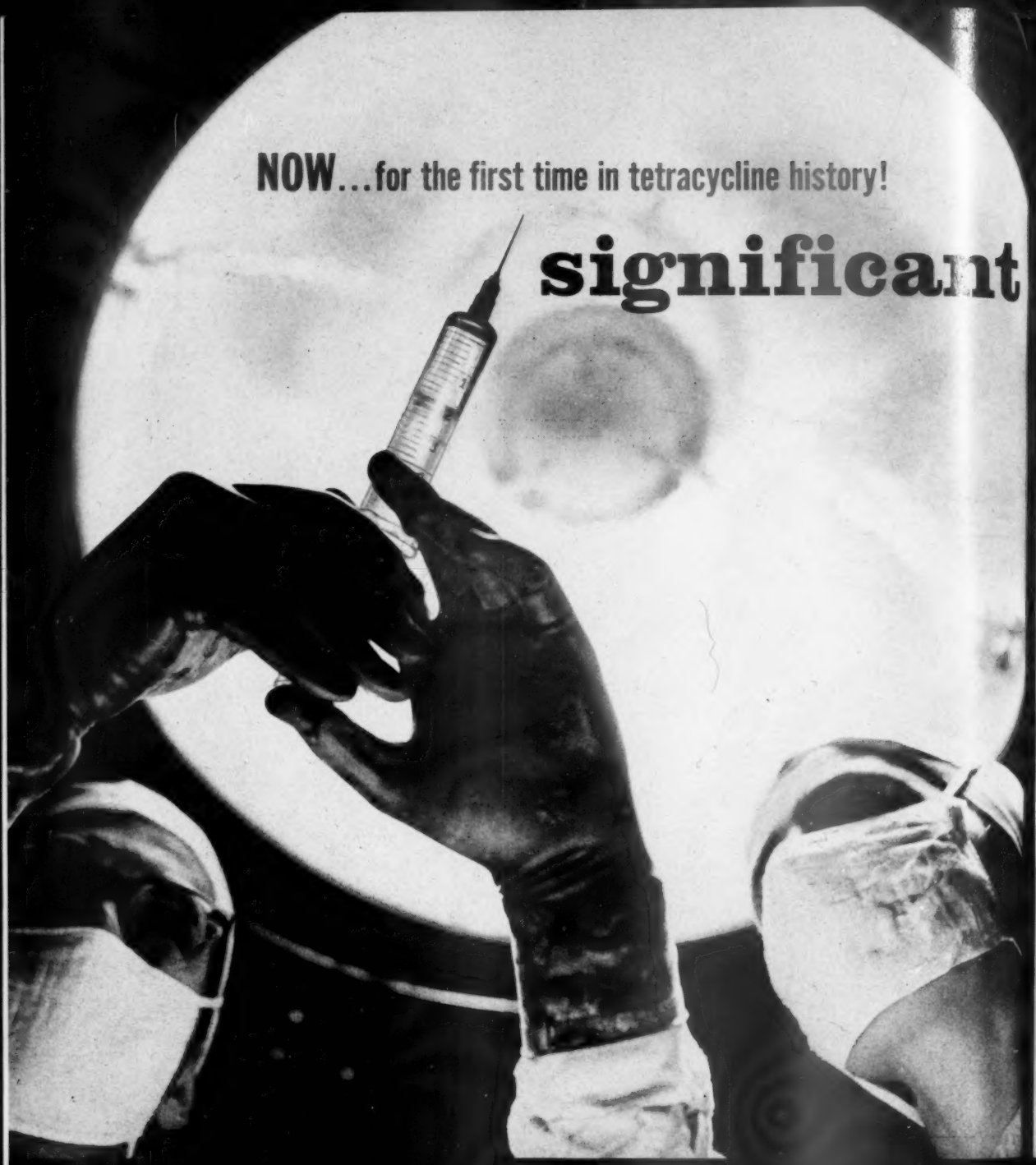

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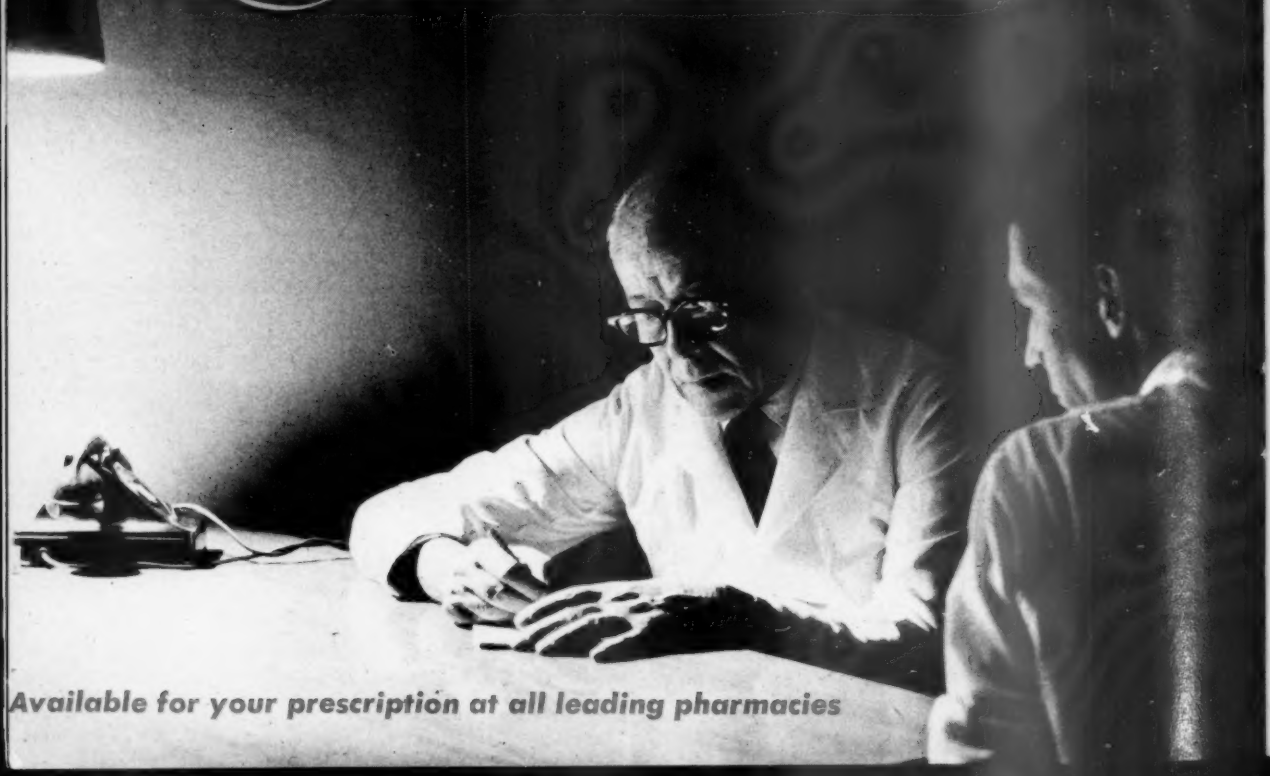

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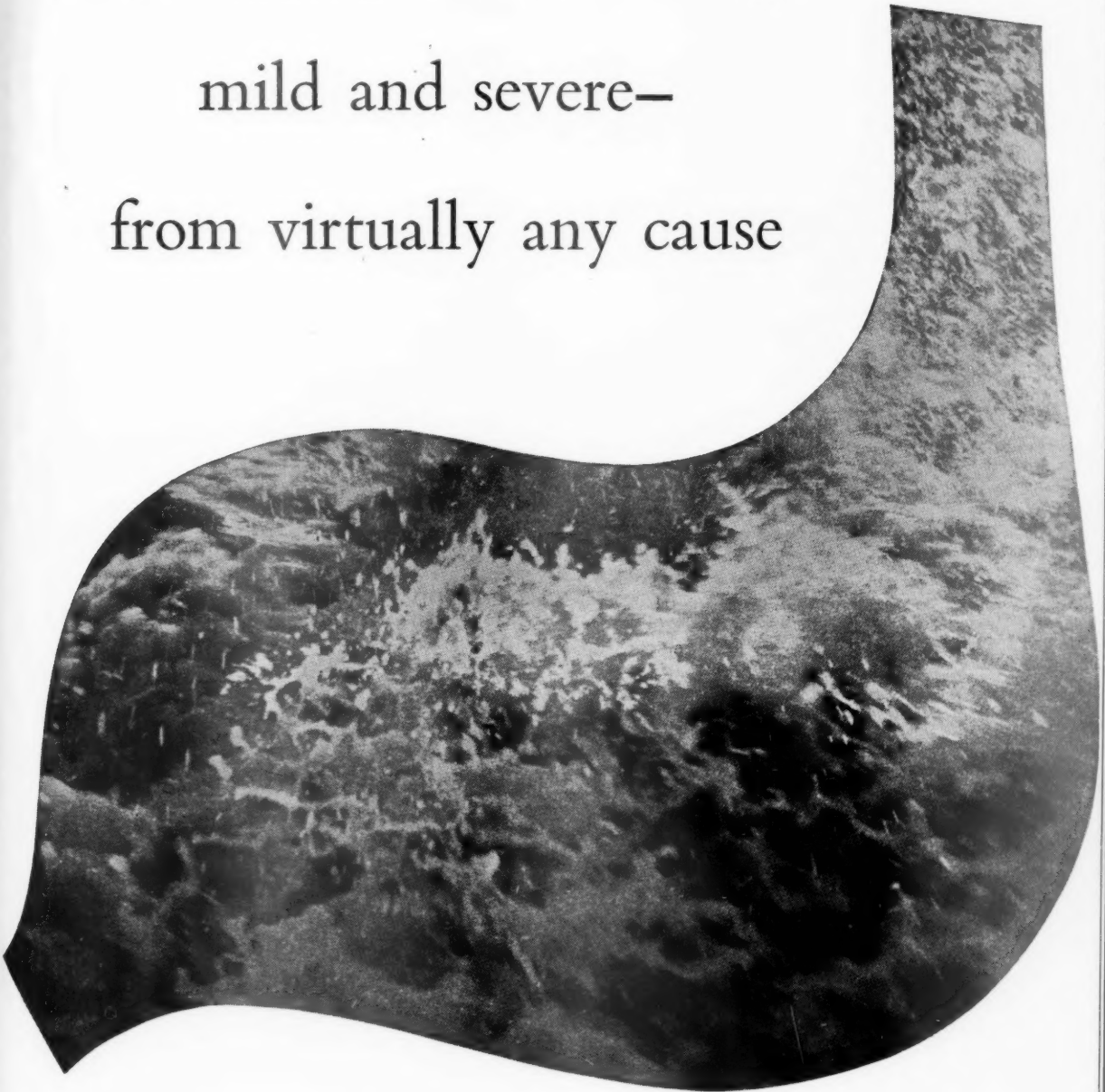

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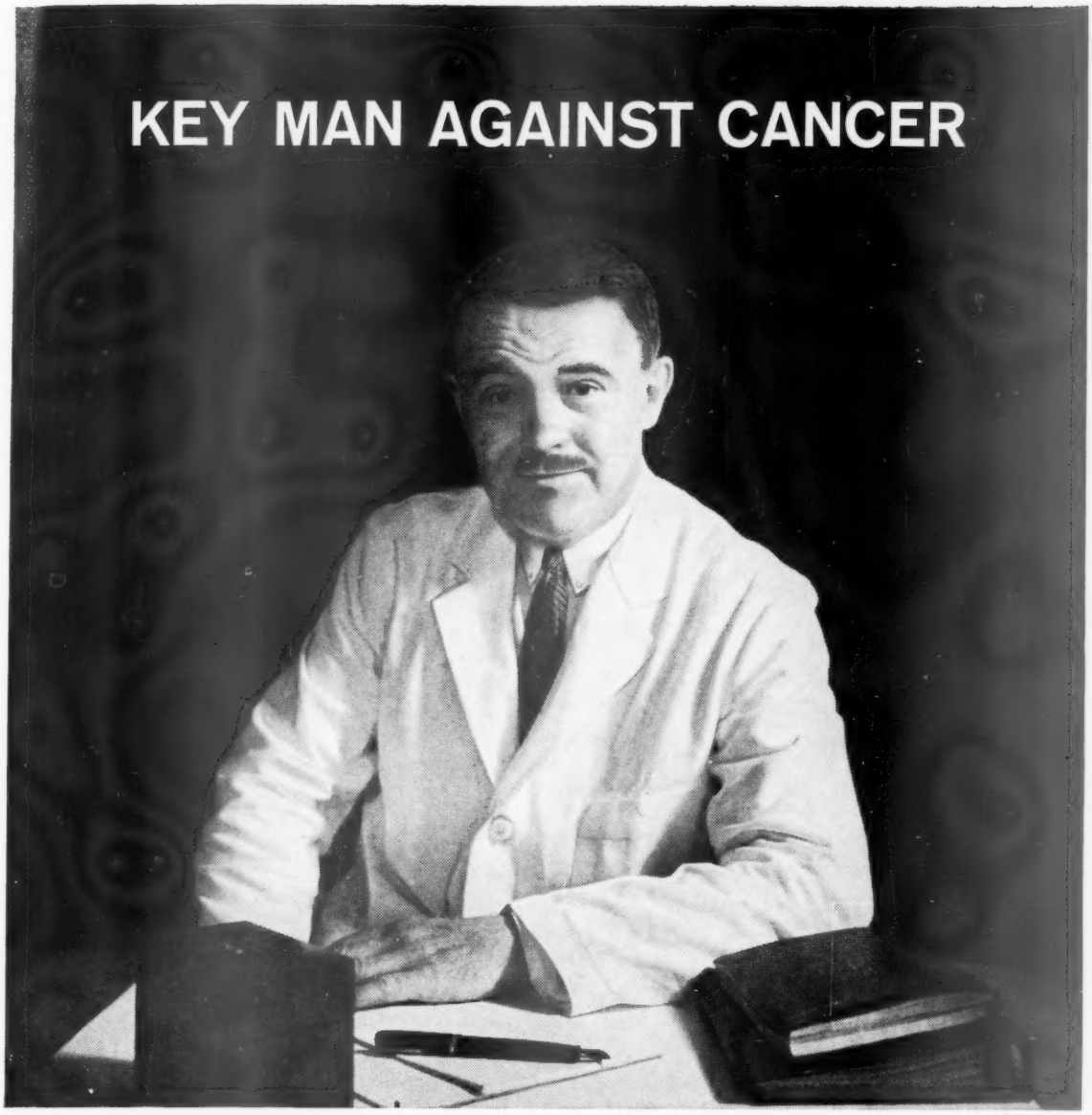

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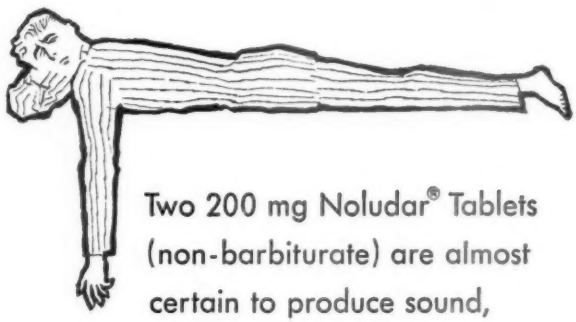
feeling 

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